THE PHILIPPINE JOURNAL OF SCIENCE

VOL. 53 JANUARY, 1934 No. 1

LEPROSY IN CEBU, II

By J. RODRIGUEZ and F. C. PLANTILLA

Of the Bureau of Health, Cebu, Cebu

GENERAL DATA

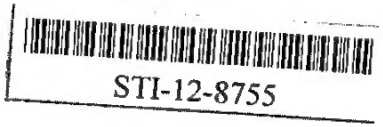
PRESENT SURVEY

In a report that appeared in 1931 [1] on the incidence of leprosy in Cebu Province written by the senior author, it was stated that a marked focal or spotted distribution of the disease existed in the province. The explanation for this was not apparent, although there was a certain correlation between density of population and prevalence of leprosy. The climate was not considered important, at least in Cebu. A brief history of the disease in this province was also given.

Since the publication of this paper, examination of contacts to recently discovered lepers has been undertaken by the Cebu Skin Dispensary, but due to the lack of personnel and, especially, of funds for transportation, this could not be done systematically.

In December, 1932, it was decided to visit systematically the homes of all newly reported "closed" and "open" cases of leprosy that could be reached on foot within the city of Cebu and its environs. At the same time, it was planned to make a thorough health survey of the barrio of Baud in the district of San Nicolas, which was the best-known focus of the disease in Cebu Island. This barrio is bounded on the north by Carlock Street, on the east by Tuti Street, on the west by C. Padilla

[1] Philip. Journ. Sci. 45 (1931) 459–481.



Street, and on the south by Pagina River. It has an area of less than half a square mile and is estimated to contain about 600 households, representing approximately 3,500 people. This was to serve as the control for the urban cases occurring in the city of Cebu. To aid in this undertaking and in order to establish a liaison with the people of the district, a branch of the dispensary was established there.

At the same time, a similar survey of the normal population was started on the outskirts of the town of Opon, which is situated on Mactan Island just across the narrow Cebu Strait from the capital. This was to provide a control for the rural cases of leprosy. It was intended to visit another 600 homes in this district.

In addition, the former homes of many of the lepers segregated during the last two years as well as the houses of some of the registered "closed" cases and paroled ex-lepers were also surveyed. As too few of the contacts of recent positive cases could be located, it was decided to include some of those segregated as long as three years previously and in a few instances even longer than this. This introduced a very grave source of error in the interpretation of some of the data covered by this preliminary report, but it was felt that the records obtained would prove useful in the future when other secondary cases in these households may be expected to develop. This subject will be discussed more fully later.

To May 15, 1933, it has been possible to secure epidemiological data on ninety-five nonleper households in the barrio of Baud. Unfortunately, the survey had to be started in what was perhaps the poorest part of the barrio, so that these control families appeared to be poorer than the average and did not seem truly representative of the households in the barrio. However, the results so far are not devoid of some interest.

On the other hand, the control rural households surveyed at Opon, of which there were 215, were possibly better off than the average, as they are generally nearer to the town than most of the leprous households visited and consequently had better chances for employment in the few industrial establishments located in Opon. Needless to say, these inequalities will tend to disappear both at Baud and at Opon as more control families are surveyed.

In most of the subsequent analyses of our data, the control and "leper" groups from both places have been combined; al-

though such a step is open to many objections, it tended to make this group more nearly representative of the normal population of Cebu Province.

However, whenever interesting or any marked differences appeared to exist between the urban and rural sets of control and "leper" households, these were also discussed or at least mentioned in the present report. For this reason, tables covering the most important data, such as the population, etc., for the Cebu (urban) and the Mactan (rural) control and "leper" families, are given separately.

POPULATION

In the city of Cebu, 193 households were surveyed of whom 95 were control families living in the barrio of Baud, 16 were households which have been exposed to a "positive" case of leprosy in the district of San Nicolas, and 16 were households similarly exposed but were located outside of San Nicolas district, although still within the city limits. Twenty-seven other households in San Nicolas had one or more incipient cases of leprosy among their members, while 39 others outside of this

TABLE 1.—*Population, by age and sex, of Cebu households surveyed.*

Age.	Controls (95 families).			Incipients (66 families).			Positives (32 families).			Total lepers (98 families).		
	M	F	Total.	M	F	Total.	M	F	Total.	M	F	Total.
Yrs.												
1—	6	5	11	6	8	14	1	1	2	7	9	16
1	9	5	14	5	1	6	4	0	4	9	1	10
2	4	3	7	5	6	11	4	3	7	9	9	18
3	8	7	15	9	2	11	1	0	1	10	2	12
4	5	2	7	11	4	15	3	0	3	14	4	18
5– 9	32	24	56	24	33	57	15	12	27	39	45	84
10–14	24	27	51	22	36	58	9	11	20	31	47	78
15–19	29	42	71	28	35	63	17	23	40	45	58	103
20–29	47	70	117	38	46	84	23	22	45	61	68	129
30–39	33	23	56	18	26	44	9	7	16	27	33	60
40–49	20	20	40	17	22	39	10	12	22	27	34	61
50–59	6	18	24	12	10	22	5	5	10	17	15	32
60–69	12	10	22	4	7	11	3	4	7	7	11	18
70–79	2	1	3	1	4	5	0	0	0	1	4	5
80–89	0	2	2	1	2	3	1	0	1	2	2	4
90+	2	0	2	0	0	0	0	1	1	0	1	1
Total...	239	259	498	201	242	443	105	101	206	306	343	649

TABLE 2.—*Population, by age and sex, of Mactan households surveyed.*

Age.	Controls (215 families).			Incipients (25 families).			Positives (56 families).		
	M	F	Total.	M	F	Total.	M	F	Total.
Yrs.									
Less than 1	46	30	76	1	3	4	3	3	6
1	16	13	29	1	0	1	0	4	4
2	19	18	37	4	1	5	6	7	13
3	32	33	65	1	3	4	3	4	7
4	22	18	40	1	1	2	3	5	8
5 to 9	117	100	217	13	6	19	29	24	53
10 to 14	80	77	157	8	15	23	18	22	40
15 to 19	68	71	139	11	11	22	18	22	40
20 to 29	130	135	265	8	12	20	29	28	57
30 to 39	71	69	140	6	7	13	8	16	24
40 to 49	36	35	71	9	5	14	17	11	28
50 to 59	21	20	41	3	8	11	7	14	21
60 to 69	9	22	31	3	2	5	4	6	10
70 to 79	3	3	6	0	3	3	5	4	9
80 to 89	2	1	3	1	2	3	1	1	2
90+	0	2	2	0	1	1	0	1	1
Total	672	647	1,319	70	80	150	151	172	323

Age.	Paroled (34 families).			Contacts (115 families).		
	M	F	Total.	M	F	Total.
Yrs.						
Less than 1	2	1	3	6	7	13
1	1	4	5	2	8	10
2	4	1	5	14	9	23
3	6	1	7	10	8	18
4	4	3	7	8	9	17
5 to 9	8	15	23	50	45	95
10 to 14	22	9	31	48	46	94
15 to 19	13	15	28	42	48	90
20 to 29	12	19	31	49	59	108
30 to 39	4	11	15	18	34	52
40 to 49	8	11	19	34	27	61
50 to 59	3	5	8	13	27	40
60 to 69	2	5	7	9	13	22
70 to 79	0	1	1	5	8	13
80 to 89	1	0	1	3	3	6
90+	0	0	0	0	2	2
Total	90	101	191	311	353	664

TABLE 3.—*Population, by age and sex, Cebu and Mactan combined.*

Age.	Controls (310 families).[a]			Incipients (91 families).[b]			Positives (88 families).[c]		
	M	F	Total.	M	F	Total.	M	F	Total.
Yrs.									
Less than 1	52	35	87	7	11	18	4	4	8
1	25	18	43	6	1	7	4	4	8
2	23	21	44	9	7	16	10	10	20
3	40	40	80	10	5	15	4	4	8
4	27	20	47	12	5	17	6	5	11
5 to 9	149	124	273	37	39	76	44	36	80
10 to 14	104	104	208	30	51	81	27	33	60
15 to 19	97	113	210	39	46	85	35	45	80
20 to 29	177	205	382	46	58	104	52	50	102
30 to 39	104	92	196	24	33	57	17	23	40
40 to 49	56	55	111	26	27	53	27	23	50
50 to 59	27	38	65	15	18	33	12	19	31
60 to 69	21	32	53	7	9	16	7	10	17
70 to 79	5	4	9	1	7	8	5	4	9
80 to 89	2	3	5	2	4	6	2	1	3
90+	2	2	4	0	1	1	0	2	2
Total	911	906	1,817	271	322	593	256	273	529

Age.	Paroled (34 families).[d]			Total lepers (213 families).[e]		
	M	F	Total.	M	F	Total.
Yrs.						
Less than 1	2	1	3	13	16	29
1	1	4	5	11	9	20
2	4	1	5	23	18	41
3	6	1	7	20	10	30
4	4	3	7	22	13	35
5 to 9	8	15	23	89	90	179
10 to 14	22	9	31	79	93	172
15 to 19	13	15	28	87	106	193
20 to 29	12	19	31	110	127	237
30 to 39	4	11	15	45	67	112
40 to 49	8	11	19	61	61	122
50 to 59	3	5	8	30	42	72
60 to 69	2	5	7	16	24	40
70 to 79	0	1	1	6	12	18
80 to 89	1	0	1	5	5	10
90+	0	0	0	0	3	3
Total	90	101	191	617	696	1,313

[a] Controls = 310 households, 1,817 persons or 5.8 persons per household.
[b] Incipients = 91 households, 593 persons or 6.5 persons per household.
[c] Positives = 88 households, 529 persons or 6.0 persons per household.
[d] Paroled = 34 households, 191 persons or 5.6 persons per household.
[e] Total lepers = 213 households, 1,313 persons or 6.2 persons per household. Total controls and lepers: 523 households, 3,130 persons or 6.0 persons per household.

TABLE 4.—*Population, by age and sex, control and leper inhabitants of Cebu and Mactan combined.*

Age.	Controls.[a]						Leper households.[b]					
	Males.		Females.		Total.		Males.		Females.		Total.	
Yrs.		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*
1—	52	5.7	35	3.9	87	4.8	13	2.1	16	2.3	29	2.2
1	25	2.7	18	2.9	43	2.4	11	1.8	9	1.3	20	1.5
2	23	2.5	21	2.3	44	2.4	23	3.7	18	2.6	41	3.1
3	40	4.4	40	4.4	80	4.4	20	3.2	10	1.4	30	2.3
4	27	3.0	20	2.2	47	2.6	22	3.6	13	1.9	35	2.7
5– 9	149	16.4	124	13.7	273	15.0	89	14.4	90	12.9	179	13.6
10–14	104	11.4	104	11.5	208	11.4	79	12.8	93	13.4	172	13.1
15–19	97	10.6	113	12.5	210	11.6	87	14.1	106	15.2	193	14.7
20–29	177	19.5	205	22.6	382	21.0	110	17.8	127	18.3	237	18.0
30–39	104	11.4	92	10.1	196	10.8	45	7.3	67	9.7	112	8.5
40–49	56	6.1	55	6.1	111	6.1	61	9.9	61	8.8	122	9.3
50–59	27	3.0	38	4.2	65	3.6	30	4.9	42	6.0	72	5.5
60–69	21	2.3	32	3.5	53	2.9	16	2.6	24	3.4	40	3.0
70–79	5	0.5	4	0.4	9	0.5	6	1.0	12	1.7	18	1.4
80–89	2	0.2	3	0.3	5	0.3	5	0.8	5	0.7	10	0.8
90+	2	0.2	2	0.2	4	0.2	0	0.0	3	0.4	3	0.2
Total...	911	99.9	906	99.9	1,817	100.0	617	100.0	696	100.0	1,313	99.9

[a] Controls: Males = 911, or 51.1 per cent; females = 906, or 49.9 per cent.
[b] Leper households: Males = 617, or 46.9 per cent; females = 696, or 53.0 per cent.

district also had incipient lepers. Summarizing, there were 95 control households and 98 "leper" households; of the latter, 66 were exposed to incipient or "closed" cases and 32 to positive cases.

The population, by age and sex, of the control and "leper" households in the city of Cebu, appears in Table 1. There were 498 individuals of both sexes in the control group and 649 in the "leper" group. These latter represent the "contacts" to the disease.

In the town of Opon, there were visited 215 control households, 25 homes with "closed" cases, 56 with positive cases, and 34 with "paroled" negative lepers, making a total of 330 households surveyed, with 1,983 inhabitants. Table 2 gives the population, by age and sex, of these households.

In Table 3 the urban and rural groups have been combined. The control households total 310 houses, with 1,817 persons or 5.8 persons per household. There were 91 households with closed cases of leprosy, representing 593 contacts among them. In 88 homes, 529 persons were exposed to a "positive" or "open" case, while 191 individuals in 34 homes were in contact with

paroled ex-lepers. Summarizing, there were 310 households with 1,817 persons in the control group and 213 households with 1,313 people in the "leper" group, making a total of 523 households surveyed, representing 3,130 people.

Table 4 gives the percentage of distribution, by age and sex, of the entire population in the two groups. Among the controls, 911, or 51.1 per cent, were males and 906, or 49.9 per cent, were females, while of the total population of 1,313 in households where cases of leprosy have appeared, 617, or 46.9 per cent, were males and 696, or 53 per cent, were females.

TABLE 5.—*Occupation, arranged in broad groups, of control and leper contact population of Mactan and Cebu combined.*

Occupation.	Controls.					
	Males.		Females.		Total.	
		P. ct.		*P. ct.*		*P. ct.*
1. Domestic	6	1.0	47	7.3	53	4.3
2. Employee	26	4.4	3	0.5	29	2.3
3. Farmer	33	5.6	0	0.0	33	2.6
4. Fisherman	64	10.8	7	1.1	71	5.7
5. Merchant	14	2.4	44	6.8	58	4.7
6. Ordinary laborer	207	34.9	8	1.2	215	17.3
7. Skilled laborer	46	7.8	34	5.2	80	6.5
8. Professional	3	0.5	1	0.2	4	0.3
9. Property owner	1	0.2	0	0.0	1	0.1
10. Student	90	15.1	68	10.5	158	12.7
11. Housework	0	0.0	387	59.7	387	31.2
12. No occupation	103	17.3	49	7.6	152	12.2
Total	593	100.0	648	100.1	1,241	99.9

Occupation.	Leper contacts.					
	Males.		Females.		Total.	
		P. ct.		*P. ct.*		*P. ct.*
1. Domestic	7	1.6	37	6.8	44	4.5
2. Employee	28	6.4	4	0.7	32	3.3
3. Farmer	38	8.7	24	4.4	62	6.3
4. Fisherman	23	5.2	0	0.0	23	2.3
5. Merchant	13	3.0	40	7.3	53	5.4
6. Ordinary laborer	130	29.6	12	2.2	142	14.4
7. Skilled laborer	32	7.3	23	4.2	55	5.6
8. Professional	2	0.4	0	0.0	2	0.2
9. Property owner	4	0.9	5	0.9	9	0.9
10. Student	71	16.2	78	14.3	149	15.1
11. Housework	0	0.0	268	49.0	268	27.1
12. No occupation	91	20.7	56	10.2	147	14.9
Total	439	100.0	547	100.0	986	100.0

In the control population, the mean age for the males was 20.45 ± 0.37 years, the standard deviation being 16.65 ± 0.26. The mean age among the females was 22.20 ± 0.38, with a standard deviation of 17.10 ± 0.27 years. The difference in the mean ages of the males and the females was 1.75 ± 0.53 in favor of the females. This difference is on the border of being significant.

Among the leper contacts, the mean age for the males was 22.70 ± 0.39, with a standard deviation of 18.0 ± 0.35; while the mean age among the females was 25.15 ± 0.48, with a standard deviation of 18.7 ± 0.34. The above figures indicate that as among the controls, the females were also older than the male population among the contacts, the difference between the two sexes being 2.45 ± 0.606 years.

The males among the leper contacts are on the average older than the males in the control group, the difference in the mean ages being 2.25 ± 0.54 years. The difference in the mean ages of the females in both groups of population was 2.95 ± 0.61, likewise in favor of the contact population.

It may be stated, therefore, that in both sets of population the mean ages of the females were higher than those of the males, while the mean ages of both sexes were higher in the contact group than in the control population.

OCCUPATION

The occupations of all inhabitants of the two groups who were 10 or more years old are given in Table 5. In two males in the control group, the occupation was not stated in our records; these do not appear in the table. There were slightly more farmers and less fishermen among the leper contacts than in the control population, less skilled or ordinary laborers, somewhat more property owners and students, but also relatively more that had no occupation. None of these differences, however, was significant.

LITERACY

It was possible to secure literacy data (Table 6) on only 1,230 persons in the control population, and 955 in the leper group. There was more illiteracy in the leper group, the difference in percentages being 9.5 ± 2.13. This difference was even more marked in the rural households surveyed. There were correspondingly fewer who could read and speak English, Spanish, or Visayan among the "contacts."

TABLE 6.—*Literacy, control population, Cebu and Mactan.*

Language.	Control group.[a]			"Leper" group.[b]		
	Males.	Females.	Total.	Males.	Females.	Total.
English and Spanish	14	5	19	12	12	24
English and Visayan	285	227	512	177	162	339
Spanish and Visayan	16	4	20	14	0	14
Visayan only	97	108	205	62	58	120
Illiterate	175	299	474	163	295	458
Total	587	643	1,230	428	527	955

[a] Control group: Illiterates, 38.5 per cent; knew English, Spanish, or both, 44.8 per cent; Visayan only, 16.7 per cent.

[b] Leper group: Illiterates, 47.9 per cent; knew English, Spanish, or both, 39.4 per cent; Visayan only, 12.7 per cent.

MORTALITY

Table 7 gives the number of deaths in the control population, as well as among the contacts to positive lepers, incipient lepers, "paroled negative" lepers, and "total lepers," since the establishment of the family.

The total mortality is lower in the control group than among the "contacts," the death rate among the former being 18.76 ± 0.8 per hundred of living population, and 26.85 ± 1.04 per cent among the latter, giving a significant difference of 8.08 ± 1.35 per cent. It must be remembered that these figures cover an indeterminable number of years and do not represent a yearly or fixed rate. In order to arrive at a more accurate comparison of the rates in the two sets of households, we have worked out the death rate in only one year, that of 1932. It was found to be 12.65 ± 2.62 per thousand among the control population and 18.28 ± 3.41 per thousand for the contacts, the difference being 5.53 ± 4.53, which is not significant. The death rate for the entire province for the corresponding year was 13.81 per thousand.

INCIDENCE OF LEPROSY

It is not possible to determine the true rate or incidence of leprosy in the present report for obvious reasons. Not until the entire barrio of Baud and the town of Opon have been surveyed will it be possible to estimate the incidence of leprosy in these districts. This inability to determine not only the gross incidence of the disease, but also the true rate by sex, by age groups, by income, etc., has naturally seriously handicapped the proper analysis of the available data in the present report.

TABLE 7.—*Mortality, controls and leper contacts, Mactan and Cebu combined.*[a]

Age.	Controls.			Total lepers.			Incipients.		
	M	F	Total.	M	F	Total.	M	F	Total.
Yrs.									
Less than 1	76	44	120	70	60	130	36	30	66
1	32	28	60	28	28	56	16	12	28
2	19	18	37	22	16	38	7	9	16
3	14	14	28	13	9	22	6	4	10
4	8	9	17	3	9	12	2	3	5
5 to 9	15	27	42	27	26	53	8	9	17
10 to 14	6	9	15	12	17	29	3	10	13
15 to 19	6	10	16	9	11	20	3	3	6
20 to 29	7	6	13	18	9	27	4	1	5
30 to 39	7	4	11	8	11	19	3	3	6
40 to 49	9	0	9	15	7	22	6	1	7
50 to 59	5	2	7	16	4	20	4	1	5
60 to 69	9	0	9	6	0	6	3	0	3
70 to 79	2	1	3	1	1	2	1	1	2
80 to 89	1	1	2	1	1	2	1	1	2
90+	1	1	2	0	0	0	0	0	0
Stillbirths	14	17	31	15	9	24	0	0	0
Total	231	191	422	264	218	482	103	88	191

Age.	Positives.			Paroled.		
	M	F	Total.	M	F	Total.
Yrs.						
Less than 1	28	22	50	6	8	14
1	12	13	25	0	3	3
2	12	4	16	3	3	6
3	6	3	9	1	2	3
4	1	6	7	0	0	0
5 to 9	16	15	31	3	2	5
10 to 14	6	6	12	3	1	4
15 to 19	4	6	10	2	2	4
20 to 29	10	7	17	4	1	5
30 to 39	3	3	6	2	5	7
40 to 49	8	5	13	1	1	2
50 to 59	9	3	12	3	0	3
60 to 69	2	0	2	1	0	1
70 to 79	0	0	0	0	0	0
80 to 89	0	0	0	0	0	0
90+	0	0	0	0	0	0
Stillbirths	13	7	20	2	2	4
Total	130	100	230	31	30	61

[a] Total deaths, controls = 422; leper contacts = 482. Total mortality controls = $422 + 1{,}817 = 2{,}239$. $\frac{422 \times 100}{2{,}239} = 18.84 \pm 0.8$ per hundred of living population.

Leper contacts = $482 + 1{,}313 = 1{,}795$. $\frac{482 \times 100}{1{,}795} = 26.85 \pm 1.04$ per hundred of living population.

Difference = 8.03 ± 1.35 per hundred of living population.

COMPARISON OF CONDITIONS IN CONTROL AND IN LEPER HOUSEHOLDS

In this section, an analysis will be made of the economic and hygienic conditions in both control and "leper" households. By "leper" households or families are meant those in which one or more definite cases of leprosy have developed since their establishment. In other words, the members of such households or families may be considered as "contacts" to the disease, with the exception of those who were born after the case or cases in the household or family had been segregated.

It must be kept in mind that the unit being compared in this section is the "household." Significant differences as to hygienic and economic conditions in the two sets of households will be pointed out, and similarities will be mentioned.

The percentages compared in this report have been tested statistically, although the calculations do not always appear in the text, for the sake of brevity. Whenever the differences are not statistically significant or are merely on the point of being so, these will be mentioned.

Every effort was made to make the data regarding the households surveyed as complete as the circumstances allowed. Directions for filling the blanks in the forms were made as explicit as possible. Four employees of the Cebu Skin Dispensary were assigned to take over the field work but one had to be dropped later. At first, these four, working as a group, were accompanied by both of us to insure uniformity of methods. Later, each worker made the visits accompanied by either one of us until we were sure that the proper procedures were being followed. None of these field workers was assigned to any particular region or to a special class of households. They had other duties to perform besides the survey, and they were sent out as opportunity offered or were shifted about if necessary. Almost all the visits were made on foot.

The work is now so organized that a visit is made to the home of a newly reported case within, at the most, a week after the diagnosis has been made, provided the home can be reached on foot. It is proposed to resurvey the homes and examine as many of the leper households as possibly every six months.

The following discussion is based on the results of the first visits made to the 310 control and 213 leper households already mentioned.

It may be objected that even if the visit to the home of a newly detected leper were made as soon as the diagnosis is arrived at, the conditions in that household at the onset and during the development of the disease might not be the same as at the time the visit was made. Furthermore, the former conditions will never be known; because, due to the long incubation period, the visit is made necessarily from two to five or more years too late. This objection is legitimate only with regard to the first recorded case, but if the visits were made to the same households regularly, say at least once every six months, and new cases developed in some of them, it would then be possible to have an idea of the conditions in these households during the development of the disease.

Although in some cases the households were visited soon after the diagnosis was arrived at, in many others the households were surveyed from one to three years after the lepers had been detected, and in a few cases at even longer intervals. Therefore, these records would seem to be quite useless in many respects; at least, in so far as the previous cases are concerned. Our present data were analyzed chiefly to orientate further work and to find weak points in our present methods of gathering and recording data.

However, the findings proved interesting enough to merit publication of the gross results. Moreover, we believe that the data reflect in a general way the differences, if any, between the control households and the homes where leprosy has occurred.

The analysis of the data covering the following items has been partially completed and will be very briefly presented here.

KIND OF HOUSE

Houses were classified as to construction as (*a*) bamboo, (*b*) mixed materials, (*c*) wooden, and (*d*) stone and wood.

The size of the house in meters and the number of rooms were determined. The kinds of floor, walls, and roof were recorded and whether the house was owned or rented by the occupant and the amount of rent, if any, were noted. The age of the house was also asked for, and the state of repair was noted. The distance to the main road was estimated, as well as proximity to the sea or a stream. Finally, the ground floor was examined and the use made of it taken down.

Table 8 shows that if the data from Cebu and Mactan are combined, 62.5 per cent of the control households lived in bamboo

houses while only 52.5 per cent of the "leper" households had such houses. Therefore, the leper families seemed to live in more substantial houses than the controls; the difference, however, is not statistically significant. In Opon, there was no difference in the two groups, both having about the same proportion of nipa houses. In Cebu, 64.2 per cent of the controls lived in nipa shacks, while only 42.7 per cent of the houses with cases of leprosy were of similar construction.

TABLE 8.—*Kind of house; Cebu and Mactan combined.*

Kind of house.	Incipients.		Positives.		Paroled.		Total controls.		Total contacts.	
		P. ct.		P. ct.		P. ct.		P. ct.		P. ct.
Bamboo	47	51.7	46	52.3	19	55.9	194	62.5	112	52.5
Mixed material	17	18.7	21	23.8	12	35.3	66	21.3	50	23.4
Wooden	15	16.5	19	21.6	3	8.8	47	15.2	37	17.4
Stone and wood	3	3.3	0	0.0	0	0.0	2	0.7	3	1.4
No data	9	9.9	2	2.3	0	0.0	1	0.3	11	5.3
Total	91	100.1	88	100.0	34	100 0	310	100.0	213	100.0

OVERCROWDING

Overcrowding in the physical sense may be due to excessive nearness of houses to one another or to lack of sufficient space within the houses themselves.

Nearness of houses to one another.—Arbitrarily, the house being surveyed was considered "very near" the other houses if it was less than 5 meters to the nearest house. If there were "very near" houses on at least three sides, it was said to be overcrowded; if only on two sides, merely "crowded." If the nearest house was from 5 to 20 meters distant, it was considered "near," while a distance of 20 to 100 meters was called "far" and farther than 100 meters, "isolated."

Table 9 shows that 97.1 per cent of the control houses were not farther than 20 meters from the nearest house, whereas 80.8 per cent of the "leper" houses were similarly situated. The difference was 16.3 ± 2.84 per cent. Therefore, there was more overcrowding of houses among the "control" than among the "leper" homes, a result not quite expected. In Mactan the difference in this respect was even more marked, as 96.2 per cent of the control houses were within 20 meters of one another, while only 69.5 per cent of the leper homes were found to be as close to the other houses.

TABLE 9.—*Distance to other houses.*

Distance to other houses.	Incipients.		Positives.		Paroled.		Total controls.		Total contacts.	
		P. ct.		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*
Overcrowded	15	16.5	9	10.2	0	0.0	57	18.4	24	11.3
Crowded	21	23.1	12	13.7	0	0.0	26	8.4	33	15.5
Very near	18	19.8	11	12.5	5	14.7	36	11.6	34	16.0
Near	27	29.7	33	37.5	21	61.8	182	58.7	81	38.0
Far	8	8.8	15	17.0	7	20.6	5	1.6	30	14.1
Isolated	2	2.2	8	9.1	1	2.9	8	1.0	11	5.1
No data	0	0.0	0	0.0	0	0.0	1	0.8	0	0.0
Total	91	100.0	88	100.0	34	100.0	310	100.0	213	100.0

TABLE 10.—*Size of yard.*

Size of yard.	Incipients.		Positives.		Paroled.		Total controls.		Total contacts.	
		P. ct.		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*
Very small	28	30.8	20	22.7	8	23.5	65	21.0	56	26.3
Small	15	16.5	12	13.7	8	23.5	49	15.8	35	16.4
Medium	21	23.1	23	26.1	8	23.5	20	6.5	52	24.4
Large	10	11.0	12	13.7	8	23.5	4	1.3	30	14.0
No fence	11	12.1	20	22.7	2	5.9	172	55.4	33	15.5
No data	6	6.6	1	1.1	0	0.0	0	0.0	7	3.3
Total	91	100.1	88	100.0	34	99.9	310	100.0	213	99.9

Size of yard.—The degree of congestion is also reflected by the size of the yards about the houses (Table 10). In our survey a yard was considered "very small" if the space not occupied by the house was less than the area covered by the house itself; "small" if it was twice the area of the house; "medium" if three to four times; and "large" if more than four times the space occupied by the house. If there was no fence, this condition was noted.

The table shows that 36.8 per cent of the control houses had "small" and "very small" yards, while in 42.7 per cent of the leper homes they were of about the same size. Therefore, more "leper" homes seemed to have smaller yards; the difference, however, is not significant statistically.

Number of rooms in house.—The extent of overcrowding within the house itself is reflected by the number of rooms or partitions in the house. In the numbers of rooms given in Table 11, all subdivisions were included, except the kitchen and the "batalan," or small closets. The "sala" and "comedor" were included.

TABLE 11.—*Number of rooms.*

Number of rooms.	Incipients.		Positives.		Paroled.		Total controls.		Total contacts.	
		P. ct.		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*
1	9	9.9	15	17.0	6	17.7	33	10.6	30	14.1
2	28	30.8	36	41.0	20	58.8	74	23.9	84	39.4
3	26	28.6	19	21.6	5	14.7	95	30.7	50	23.4
4	10	11.0	11	12.5	2	5.9	54	17.4	23	10.8
5	3	3.3	1	1.1	0	0.0	23	7.4	4	1.9
6	3	3.3	1	1.1	0	0.0	7	2.3	4	1.9
7	0	0.0	0	0.0	0	0.0	2	0.6	0	0.0
8	1	1.1	1	1.1	0	0.0	1	0.3	2	0.9
No data	11	12.1	4	4.6	1	2.9	21	6.8	16	7.5
Total	91	100.1	88	100.0	34	100.0	310	100.0	213	99.9

TABLE 12.—*Unit space per adult person.*

Unit space per person.	Incipients.		Positives.		Paroled.		Total controls.		Total contacts.	
Sq. m.		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*
0 to 3	38	41.8	32	36.3	16	47.0	70	22.6	86	40.3
4 to 7	33	36.3	32	36.3	14	41.4	124	40.0	79	37.0
8 to 11	7	7.7	6	6.8	2	5.9	55	17.7	15	7.2
12 to 15	4	4.4	6	6.8	1	2.9	27	8.7	11	5.2
15+	6	6.6	12	13.7	1	2.9	34	11.0	19	8.9
No data	3	3.3	0	0.0	0	0.0	0	0.0	3	1.4
Total	91	100.1	88	99.9	34	100.0	310	100.0	213	100.0

The table shows that 34.5 per cent of the control homes had less than three rooms, while 53.5 per cent of the leper houses had less than this number. This shows that in spite of the fact that the houses where leprosy had developed were built of better material than the control homes, they had fewer rooms and that there was more chances of contact between the members of the household.

Unit space per person.—Finally, the best way to determine the degree of overcrowding within the house would be to determine the amount of space available for each person. To obtain these data, we measured the area of occupied space in the house (excluding lean-tos, storerooms, etc.), and divided that by the number of persons in the household, taking each individual 10 years or older as one unit and those below this age as one-half. The result then is the amount of space in square meters per adult person in the household. Table 12 gives these data for Cebu and Mactan combined.

It will be noted that in 37.4 per cent of the control households, the members had 8 square meters or more space per person, while only 21.3 per cent of the leper homes had as much space per person. The difference is significant statistically, being 16.1 ± 3.94 per cent. Therefore, there was clearly more overcrowding in the latter homes than in the controls. The results were the same in the urban (Baud) and the rural (Opon) groups.

As the question of overcrowding is obviously important in leprosy, we have endeavored to check the above results by analyzing our available data in a somewhat different manner, using individuals as units rather than households. The total population of the combined leper and control households was ascertained and then subdivided according to the unit space occupied per person. The relative incidence of leprosy was then determined for the various subdivisions. It must be emphasized that the rates thus obtained are useful merely for comparative purposes and should not be considered as the true or actual rates since they are not based on the total population of a given area or district.

TABLE 13.—*Influence of overcrowding on incidence of leprosy.*[a]

Unit space per person.	Population surveyed.	Cases of leprosy.	Rate per 1,000.[b]
Sq. m.			
0 to 3	1,165	122	104.7
4 to 7	1,175	107	91.1
8 to 11	380	19	50.0
12 to 15	156	7	44.8
15+	233	15	64.4
No data	21	0	

[a] These are not true rates, as the population given above is only part of the total population of the area surveyed.

[b] Rate per 1,000 based on population surveyed so far.

Table 13 shows a decided tendency for the incidence of the disease to decrease with an increase in the number of unit spaces per person. The only exception is the last group, in which each of the 233 individuals concerned had more than 15 square meters of available floor space in his home. The reason for this exceptional group is not clear. Many of these individuals lived in small families, hence there was plenty of floor space per individual yet the incidence is higher than that in the group having only 8 to 11 square meters. This exception perhaps

indicates that there are very probably important factors other than overcrowding that influence the occurrence of leprosy in certain households.

Summarizing the differences found in housing conditions among the normal and the "leper" households, we have found that the latter were as a rule housed in better homes, which are not as crowded together as the controls, but that there was more overcrowding within the houses themselves. It should be mentioned in this connection that only when the houses were the same ones lived in by the lepers were they included in the present survey. If the house had been greatly modified since the leper lived in it or since the beginning of his disease, it was not included. Therefore, the objection that conditions during the development of the disease may be entirely different from the conditions at the time of the survey does not hold so much in this matter of the housing.

AMOUNT OF VEGETATION

It is of interest to know something about the amount of vegetation in the yard about the houses, for if the causative organism in leprosy thrives in the soil, as is suggested by some workers, the amount of shade about the house necessarily exerts some influence on the spread of the disease.

Table 14 indicates that 30.6 per cent of the control houses had little or no vegetation, while 41.8 per cent of the leper homes similarly had scanty vegetation about their homes. This difference is barely statistically significant. In determining the amount of vegetation, the instructions to the field workers were that when there was hardly any vegetation in the yard, it was to be classified as "scanty." When the shadow of the trees and

TABLE 14.—*Amount of vegetation in the yard.*

Vegetation.	Incipients.		Positives.		Paroled.		Total controls.		Total contacts.	
		P. ct.		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*
None	25	27 5	10	11.3	1	2.9	69	22.2	36	16.9
Scanty	22	24.2	22	25.0	9	26 5	26	8.4	53	24.9
Slight	28	30.8	39	44.3	22	64.7	130	41.9	89	41.8
Dense	8	8.8	14	15.9	2	5.9	80	25.8	24	11.3
Very dense	2	2.2	0	0.0	0	0.0	1	0.3	2	0.9
No data	6	6.6	3	3.4	0	0.0	4	1.3	9	4.2
Total	91	100.1	88	99.9	34	100.0	310	99.9	213	99.9

TABLE 15.—*Number of banana hills and kamungay trees in the yard.*

Bananas in yard.[a]					Kamungay in yard.[b]				
Hills.	Control households.		Leper households.		Trees.	Control households.		Leper households.	
		P. ct.		P. ct.			P. ct.		P. ct.
1 to 4	50	17.5	42	21.9	1 to 4	77	26.6	77	39.4
5 to 9	50	17.5	29	15.1	5 to 9	40	13.8	29	14.8
10 to 14	22	7.6	17	8.9	10 to 14	17	5.9	10	5.1
15+	30	10.4	25	13.0	15+	29	10.0	9	4.6
No bananas	135	47.0	79	41.0	No trees	127	43.7	71	36.2
Total	287	100.0	192	99.9	Total	290	100.0	196	100.1

[a] Data on number of banana hills were not available for 23 control and 21 leper households.
[b] Data on kamungay trees not available for 20 control and 17 leper households.

TABLE 16.—*Sewage-disposal rating.*

Sewage-disposal rating.	Incipients.		Positives.		Paroled.		Total controls.		Total contacts.	
P. ct.		P. ct.		P. ct.		P. ct.		P. ct.		P. ct.
Less than 20	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
20 to 29	1	1.1	0	0.0	0	0.0	0	0.0	1	0.5
30 to 39	8	8.8	11	12.5	9	26.5	19	6.1	28	13.1
40 to 49	22	24.2	31	35.2	20	58.8	183	59.0	73	34.3
50 to 59	9	9.9	8	9.1	4	11.8	43	13.8	21	9.9
60 to 69	20	22.0	16	18.2	0	0.0	7	2.3	36	16.9
70 to 79	15	16.5	9	10.2	0	0.0	30	9.7	24	11.3
80 +	16	17.6	13	14.8	1	2.9	28	9.0	30	14.1
Total	91	100.1	88	100.0	34	100.0	310	99.9	213	100.1

plants about the house at noon covered up to one-fourth of the yard, it was called "slight," "dense" when the shadow covered from one-fourth to three-fourths of the yard, and "very dense" if more than three-fourths of the yard was covered.

Leper homes, if our data mean anything at all, had less vegetation about them than the controls. This finding would hardly be expected if the leprosy germ thrives in the soil and if it is taken for granted that much sunlight is inimical to its growth.

Having observed that the vegetation about many of the houses often included banana and kamungay trees (*Moringa oleifera* Lam.) we decided to obtain data regarding the number of these trees about each house and the consumption of the produce by the household. The young leaves of the kamungay, or horse-radish tree, are universally used as a vegetable by the native people of Cebu and will be discussed more fully under diet.

Each hill of fruit-bearing banana plant was considered as one tree. A kamungay tree was considered "large" when the diameter of the trunk was about 3 or more inches, and "small" when it was less than this. In tabulating, two "small" trees were considered equivalent to one "large" tree.

With regard to the use of the produce, the information obtained as to what proportion was consumed by the household and how much was sold to outsiders was so confusing that it was decided not to discuss it in the present report.

Table 15 gives the number of banana hills and adult kamungay trees in the yard. It is seen that there was practically no difference between the control and the leper households with regard to the number of banana hills. More of the control households (43.7 per cent) than leper homes (36.2 per cent) seemed to have lacked even one kamungay tree, but the difference, 7.5 ± 2.94 per cent, is not statistically significant.

There were no significant differences, therefore, between the leper and the control homes with regard to the number of kamungay and banana trees in the yards.

SANITARY CONDITIONS

Sewage disposal.—Much difficulty was encountered in arriving at a rating for sewage disposal in many households, especially at Opon, because there were no toilets of any kind. In many cases, the older members of the household simply used swamps or the seashore when these were conveniently close by, or any vacant lot was utilized for the purpose. The children usually defecated in the yard about the house, even if there were surface privies or Antipolo toilets available.

Means of sewage disposal were classified into (*a*) water closets, (*b*) Antipolo system, (*c*) pit system and surface privy, (*d*) defecating anywhere in the yard, and (*e*) defecating in the sea or a stream. The possibility of pollution of the drinking water, the soil, foodstuffs, and contamination by flies, was considered in each case. The system of waste disposal was given a maximum under each of the above headings, and the total was taken as the sewage-disposal rating.

It is seen in Table 16 that 65.1 per cent of the control households had less than a rating of 50 per cent while only 47.9 per cent of the leper homes had as poor methods of waste disposal. Therefore, the leper homes were better off in the matter of sewage disposal than the controls. This difference in favor

of the leper households was particularly marked at Opon; conditions in this respect were more nearly similar in the two sets of households in the city of Cebu.

Water supply.—The different sources of water for drinking and household purposes are given below, together with the maximum and minimum percentages allowed for each, unless there was obvious indication of contamination or other sources of error.

	Per cent.
1. Rain	60–70
2. Artesian water	50–70
3. Spring	30–70
4. Water system	25–70
5. Deep well (30 feet or more deep)	25–70
6. Shallow well (less than 30 feet deep)	10–30
7. Stream (contamination not obvious)	10–20
8. Stream (obvious contamination)	0–10

In estimating the final water supply rating, the rate for the source was given a weight of 70 per cent, while the container and handling was allowed 30 per cent.

Table 17 indicates that fully 65.2 per cent of the controls had less than a rate of 50 per cent while but 25.1 per cent of the lepers had the same rating. Therefore, as a general rule, the water supply of the leper households was of better quality than that of the controls. This finding was observed in both the rural and the urban groups.

TABLE 17.—*Water-supply rating.*

Water-supply rating.	Incipients.		Positives.		Paroled.		Total controls.		Total contacts.	
P. ct.		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*
Less than 20	1	1.1	2	2.3	3	8.8	1	0.3	6	2.8
20 to 29	1	1.1	14	15.9	6	17.7	62	20.0	21	9.9
30 to 39	9	9.9	8	9.1	5	14.7	110	35.5	22	10.3
40 to 49	4	4.4	6	6.8	3	8.8	29	9.4	13	6.1
50 to 59	9	9.9	8	9.1	7	20.6	3	1.0	24	11.3
60 to 69	17	18.7	17	19.3	6	17.7	13	4.2	40	18.8
70 to 79	20	22.0	17	19.3	1	2.9	39	12.6	38	17.8
80+	30	33.0	16	18.2	3	8.8	53	17.1	49	23.0
Total.	91	100.1	88	100.0	34	100.0	310	100.1	213	100.0

Sanitary control.—The rate was based on the number of mice, bedbugs, flies, cockroaches, and mosquitoes found in the house, as well as the number of loose animals, as pigs, chickens, goats,

dogs, etc., about the house. This rating was found to be exceptionally difficult, or practically impossible, to calculate because of the time necessary to confirm the presence of bedbugs, mice, etc., and the reluctance of the people to allow the field workers to examine their larders for cockroaches, and mats and pillows for bedbugs, etc. In most of the cases, the workers had to depend largely on statements voluntarily supplied by the housewife herself.

TABLE 18.—*Sanitary-control rating.*

Sanitary-control rating.	Incipients.		Positives.		Paroled.		Total controls.		Total contacts.	
P. ct.		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*
Less than 20	0	0.0	0	0.0	0	0.0	6	2.0	0	0.0
20 to 29	2	2.2	1	1.1	0	0.0	8	2.6	3	1.4
30 to 39	4	4.4	11	12.5	7	20.3	50	16.1	22	10.3
40 to 49	30	33.0	22	25.0	9	26.7	71	22.9	61	28.6
50 to 59	30	33.0	25	28.4	9	26.7	89	28.7	64	30.0
60 to 69	20	22.0	20	22.7	7	20.3	65	21.0	47	22.1
70 to 79	5	5.5	8	9.1	2	5.9	17	5.5	15	7.0
80+	0	0.0	1	1.1	0	0.0	4	1.3	1	0.5
Total	91	100.1	88	99.9	34	99.9	310	100.1	213	99.9

TABLE 19.—*Cleanliness rating.*

Cleanliness rating.	Incipients.		Positives.		Paroled.		Total controls.		Total contacts.	
P. ct.		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*
Less than 20	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
20 to 29	0	0.0	1	1.1	0	0.0	1	0.3	1	0.5
30 to 39	8	8.8	8	9.1	7	20.3	5	1.6	23	10.8
40 to 49	12	13.2	19	21.6	9	26.7	40	12.9	40	18.8
50 to 59	13	14.3	18	20.5	9	26.7	111	35.8	40	18.8
60 to 69	23	25.3	22	25.0	7	20.3	87	28.0	52	24.4
70 to 79	23	25.3	12	13.7	2	5.9	58	18.7	37	17.4
80+	12	13.2	7	7.9	0	0.0	8	2.6	19	8.9
No data	0	0.0	1	1.1	0	0.0	0	0.0	1	0.5
Total	91	100.1	88	100.0	34	99.9	310	99.9	213	100.1

According to Table 18, the control households had a rating of less than 50 per cent in 43.6 per cent of the cases, while 40.3 per cent of the leper homes had a rating as poor as this. There seems to be a slight advantage in favor of the leper households, but the difference is not significant.

Cleanliness.—This rate depends on the cleanliness of the home and of the individual members of the household. The weights

given to the different items entering into the determination of this rate were as follows:

	Maximum per cent.
Living and bed rooms	25
Dining room and kitchen	25
Yard	25
Personal cleanliness	25

The total was taken as the rating for cleanliness. It is demonstrated in Table 19 that whereas only 14.8 per cent of the controls had a rating of less than 50 per cent, 30.1 per cent of the leper household had such a rating. This indicates that more of the households where lepers had previously lived or were living were dirty compared to the normal controls. This observation is quite remarkable in view of the fact that the majority of the leper homes were built with better and stronger materials, and as we shall mention later, the average earning capacity in both groups is about the same.

General sanitary rating.—In their epidemiologic study on pellagra in the southern portion of the United States, Goldberger, Wheeler, and Sydenstriker, aided by Barber,[2] used the general sanitary rating devised by Frost in his extensive epidemiologic survey of the Mississippi Valley some years previously, to determine the sanitary condition of the households surveyed. As he was investigating chiefly for acute waterborne infections, Frost naturally gave preponderating weight to the purity of the water supply. We believe that in the study of a disease like leprosy such a standard of general sanitary rating is not applicable.

In the absence of better knowledge regarding the transmission of the disease, we have simply taken the average of the ratings for sewage disposal, water supply, sanitary control, and cleanliness, and considered this as the general sanitary rating, thereby giving equal weight to these four sanitary items.

The result is shown in Table 20 where it is seen that while only 8.1 per cent of the control households have a general sanitary rating of 70 per cent or better, 13.6 per cent of the leper homes have a corresponding rating, indicating that the latter have a better sanitary average than the former. This result is to be expected in view of the advantage shown by the leper

[2] Public Health Reports 35 (1920) 1701–1714.

homes with regard to sewage disposal, water supply, and sanitary control, although the advantage is decidedly on the side of the controls in the matter of home and personal cleanliness.

These observations suggest that in adopting a standard for general sanitary rating in the epidemiologic study of leprosy, more weight should be given to cleanliness than to the other items, such as waste disposal, water supply, and perhaps even sanitary control, although the last needs to be further investigated.

TABLE 20.—*General sanitary rating.*

General-sanitary rating.	Incipients.		Positives.		Paroled.		Total controls.		Total contacts.	
P. ct.		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*
Less than 20	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
20 to 29	1	1.1	0	0.0	0	0.0	0	0.0	1	0.5
30 to 39	0	0.0	6	6.8	6	17.7	31	10.0	12	5.6
40 to 49	18	19.8	25	28.4	14	41.4	133	42.9	57	26.8
50 to 59	26	28.6	27	30.7	11	32.4	67	21.6	64	30.0
60 to 69	30	33.0	18	20.5	2	5.9	54	17.4	50	23.4
70 to 79	15	16.5	9	10.2	1	2.9	21	6.8	25	11.7
80+	1	1.1	3	3.4	0	0.0	4	1.3	4	1.9
Total	91	100.1	88	100.0	34	100.0	310	100.0	213	99.9

TABLE 21.—*Presence and size of pusali.*

Pusali.	Incipients.		Positives.		Paroled.		Total controls.		Total contacts.	
		P. ct.		*P. ct.*		*P. ct.*		*P. ct.*		*P. ct.*
Very small	23	25.3	15	17.0	2	5.9	33	10.6	40	18.8
Small	19	20.9	18	20.5	6	17.7	16	5.1	43	20.2
Large	9	9.9	4	4.6	1	2.9	7	2.3	14	6.6
None	38	41.8	50	56.8	25	73.5	254	82.0	113	53.0
No data	2	2.2	1	1.1	0	0.0	0	0.0	3	1.4
Total	91	100.1	88	100.0	34	100.0	310	100.0	213	100.0

TABLE 22.—*Use of soap.*

Frequency.	Control households.		Leper households.		Total.
		P. ct.		*P. ct.*	
Always	173	56.7	112	52.6	285
Usually	37	12.1	18	8.5	55
Sometimes	89	29.2	78	36.6	167
Never	6	2.0	5	2.3	11
Total	305	100.0	213	100.0	518

TABLE 23.—*Frequency of bathing among adults.*

Frequency.	Control households.		Leper households.		Total.
		P. ct.		*P. ct.*	
Daily	23	7.5	16	7.5	39
Almost daily	122	40.0	88	41.3	210
Twice a week	149	48.9	57	26.8	206
Less than twice a week	11	3.6	52	24.4	63
Total	305	100.0	213	100.0	518

TABLE 24.—*Frequency of bathing among children.*

Frequency.	Control households.		Leper households.		Total.
		P. ct.		*P. ct.*	
Daily	26	10.5	29	15.3	55
Almost daily	107	43.3	83	43.6	190
Twice a week	105	42.5	36	19.0	141
Less than twice a week	9	3.6	42	22.1	51
Total	247	99.9	190	100.0	437

Pusali.—An indication of the cleanliness or proper health habits of a household in the Philippines is the presence or absence, and if present, the size and condition, of what is known as "pusali" among Tagalogs and "panghugasan" among Cebuanos. In many Filipino homes, there is a small extension of the kitchen known in many parts of the Islands as "batalan" and called locally "pantao." Usually, it is not roofed and the flooring is of bamboo or of young saplings set apart, so as to allow better drainage and to facilitate drying. All the dish washing and scouring of pots, as well as the washing of the feet and hands, is done in the batalan. In clean homes the wash water, which accumulates under the batalan, is drained away or a pile of stones is built underneath in order to prevent chickens from scratching up the wet soil and loose pigs from wallowing there during the hot part of the day. Unless care is thus taken, the place is converted into a dirty mudhole, which is the "pusali," and in very careless households, the stench is perceptible even to the casual visitor in the "sala," or receiving room.

Table 21 shows that in 82.0 per cent of the control homes, there was no pusali, while only 53.0 per cent of leper households

did not have it. This finding again would tend to show that leper homes are less cleanly than the controls.

Frequency of bathing and the use of soap.—Data regarding these points are given in Tables 22, 23, and 24. It should be remembered that the figures appearing in these tables represent the average frequency of bathing and use of soap for the family or household as a whole and not that of individuals. They are based on the statements of the informant in each household, the only check being the general appearance of the members of the household.

TABLE 25.—*Number of persons in household (size of family).*

Persons.	Incipients.		Positives.		Paroled.		Total controls.		Total contacts.	
		P. ct.		P. ct.		P. ct.		P. ct.		P. ct.
1 to 2	1	1.1	9	10.2	2	5.9	32	10.3	12	5.6
3 to 4	14	15.4	23	26.1	8	23.6	84	27.1	45	21.1
5 to 6	26	28.6	16	18.2	9	26.4	76	24.5	51	23.9
7 to 8	17	18.7	25	28.4	6	17.4	61	19.7	48	22.5
9 to 10	20	22.0	9	10.2	8	23.6	36	11.6	37	17.4
11 to 12	9	9.9	4	4.5	1	2.9	13	4.2	14	6.6
13 to 14	1	1.1	1	1.1	0	0.0	6	1.9	2	0.9
15 to 16	2	2.2	1	1.1	0	0.0	1	0.3	3	1.4
17 to 18	1	1.1	0	0.0	0	0.0	1	0.3	1	0.5
19+	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
Total	91	100.1	88	99.9	34	100.0	310	99.9	213	99.9

TABLE 26a.—*Classification of total income of control and leper households by "adult male units."*

Income per adult male unit.	Households.									
	Incipients.		Positives.		Paroled.		Total controls		Total contacts.	
Pesos.		P. ct.		P. ct.		P. ct.		P. ct.		P. ct.
Less than 20	5	5.5	12	13.7	10	29.4	13	4.2	27	12.7
20 to 39	18	19.8	19	21.6	11	32.3	51	16.5	48	22.5
40 to 59	11	12.1	16	18.2	3	8.8	56	18.1	30	14.1
60 to 79	10	11.0	6	6.8	2	5.9	44	14.2	18	8.4
80 to 99	7	7.7	6	6.8	5	14.7	43	13.8	18	8.4
100 to 119	7	7.7	8	9.1	3	8.9	25	8.0	18	8.4
120 to 139	8	8.8	3	3.4	0	0.0	21	6.8	11	5.2
140 to 159	11	12.1	3	3.4	0	0.0	19	6.1	14	6.6
160 to 179	1	1.1	2	2.3	0	0.0	8	2.6	3	1.4
180 to 199	3	3.3	1	1.1	0	0.0	8	2.6	4	1.9
200+	10	11.0	12	13.6	0	0.0	22	7.1	22	10.3
Total	91	100.1	88	100.0	34	100.0	310	100.0	213	99.9

TABLE 26b.—*Comparative rate of leprosy per thousand of population classified according to income based on "adult male units."*[a]

Income per adult male unit.	Population surveyed.	Lepers.	Rate per 1,000.[b]
Pesos.			
Less than 40	848	117	137.9
40 to 79	970	56	57.7
80 to 119	573	41	71.5
120 to 159	345	25	72 4
160 to 199	140	9	61.4
200+	254	22	86.6

[a] This is not the true rate as the population given above is only that of a small portion of the area to be surveyed.

[b] Rate per thousand based on population surveyed.

TABLE 27.—*Regularity of rice, corn, and a mixture of both in the main diet of control and leper households.*

Presence at table.	Rice.				Corn.			
	Control households.		Leper households.		Control households.		Leper households.	
		P. ct.		*P. ct.*		*P. ct.*		*P. ct.*
Never	14	4.5	7	3.3	16	5.2	18	8.5
Very seldom	102	32.9	16	7.5	4	1.3	3	1.4
Seldom	128	38.7	127	59.6	35	11.1	46	21.6
Often	58	18.7	62	29.1	58	18.7	128	60.0
Frequently	5	1.6	0	0.0	3	1.0	18	8.5
Regularly	11	3.5	1	0.5	194	62.6	0	0.0
Total	318	99.9	213	100.0	310	99.9	213	100.0

Presence at table.	Mixture of rice and corn.			
	Control households.		Leper households.	
		P. ct.		*P. ct.*
Never	99	32.0	55	25.8
Very seldom	83	26.8	12	5.6
Seldom	76	24.5	126	59.1
Often	35	11.3	20	9.4
Frequently	7	2.2	0	0.0
Regularly	10	3.2	0	0.0
Total	310	100.0	213	99.9

The only striking observation brought out in Tables 23 and 24 is that whereas only 3.6 per cent of the control households admit that the adult members take a bath less often than twice a week, 24.4 per cent of the leper homes admit such extreme infrequency of bathing, while among the children, the corre-

sponding figures are 3.6 per cent and 22.1 per cent, respectively. These differences are statistically significant. In the use of soap, there were no differences, as can be noted in Table 22.

SIZE OF THE FAMILY

It has already been shown that on the average, the households where leprosy has occurred were larger than the controls. This is also demonstrated in Table 25, which indicates that only 18.3 per cent of the control households had more than eight persons while 26.8 per cent of the leper households had that number.

FAMILY INCOME

It must be admitted that our data on family income were based chiefly on the statements of the most responsible member of the household, but this was carefully checked by inquiring for the amount of property and other taxes paid, and especially at Opon, by estimating the amount of produce that might be expected from their land. A schedule was drawn up with the help of the municipal president, in which the average yield of corn, sweet potatoes, cassava, maguey, etc., per year per hectare of land was estimated, and the minimum production of coco, banana, and other fruit trees, etc., per year per plant was also arrived at. An arbitrary yearly income was also charged per head of productive cattle, pigs, goats, turkeys, and chickens owned by the family. We believe that the figures set down were not very far from the real family income, which in many cases the owners did not know themselves.

In epidemiologic studies where data on the family income are required, difficulty is always met in classifying the income. In the first place, it is necessary to consider the size of the family or household. Of two poor families that receive about the same income more suffering and unfavorable hygienic conditions are likely to be found in the larger family. Of course, the expenditure of a family composed chiefly of males differs somewhat from one in which the majority are females. Also, much depends on the manner in which the money is spent; whereas one family may be able to live decently and may even save a little on a small income, another of the same size and composition may be practically on the border line of starvation earning the same amount.

In order to overcome the differences in the age and sex constitution of unequal family groups, Goldberger, Wheeler, and

Sydenstriker(3), in their epidemiologic studies on pellagra already referred to, decided to—

> . . . employ a common denominator to which the individuals of both sexes and of all ages could be reduced in order to obtain a more accurately representative method of expressing the relative size of the families to be compared.
>
> In the absence of a better common denominator for this purpose, the Atwater (1915) scale of food requirements was employed, and the size of each family was computed according to this scale and expressed in terms of "adult male units". The assumption in the use of this scale was that the expenditures for total maintenance for individuals varied according to sex and age in the same proportion as did their food requirements. The assumption is by no means as accurate as could be desired; in its favor, however, it may be said that since family expenditures in the great majority of cases equaled total family income, and since food expenditures were nearly half (among poorer families considerably more than half) of total expenditures, a scale based on food requirements alone is obviously very much more accurate than one omitting any consideration whatsoever of the number, sex, and age of the individuals composing the families to be compared with respect to income. For the present purpose, therefore, the total income of each family . . . has been divided by the number of "adult male units" subsisting on the family income, and the resulting figure has been termed the "family income per adult male unit."

The scale used by them, based on the Atwater scale, was as follows:

Age.	Equivalent adult male unit.	
	Male.	Female.
Years.		
Adult (over 16)	1.0	0.8
15 to 16	0.9	0.8
13 to 14	0.8	0.7
12	0.7	0.6
10 to 11	0.6	0.6
6 to 9	0.5	0.5
2 to 5	0.4	0.4
Under 2	0.3	0.3

We adopted the same method in our survey and the results are shown in Table 26*a*. It is seen in this table that the income of the control and that of the leper families differ in certain significant details, but these differences are not as great as they may seem. This will be appreciated if the data are simplified as follows:

Income per adult male unit.	Control families.	"Leper" families.
	Per cent.	*Per cent.*
Incomes less than 100 pesos per unit	66.8	66.1
Incomes more than 100 pesos per unit	33.2	33.8

However, it is apparent in Table 26*a* that a significantly larger proportion of "leper" or "contact" households earned less than 40 pesos per adult male unit per year, than the controls (difference = 12.5 ± 3.96 per cent).

We have attempted also to analyze the data on the income by using the individuals rather than the households as units. In other words, the total population of the control and of the leper households were combined and then grouped according to the income expressed in adult male units of the families to which they belonged, and the incidence of leprosy in these groups was determined. The result is given in Table 26*b*.

Table 26*b* shows no tendency for the incidence of the disease to diminish with increase in the income, although the incidence is higher in the group with the lowest income per unit than in any of the other groups. It should be remembered that the rates recorded in Table 26*b* are not actual or real rates of the disease, because they are not based on the total population of a given district. They are useful merely for comparative purposes.

It may be adduced from the data discussed very briefly in the preceding pages that families in which leprosy had developed in Cebu Province when compared with nonleper households belonging to approximately the same social and economic status, lived in comparatively better houses, had as good or even better means of sewage disposal and safer water supply, but were larger, more overcrowded within their homes, and less cleanly in their personal habits and surroundings, than the control families.

It is important to remember, however, that both the control and the leper households belong to the poorer or possibly the poorest classes in Cebu Province. When the population of the province is taken as a whole, the disease will be found to attack chiefly the poorest households.

Attention is also called to the fact that in age and sex distribution, as well as in occupation and family income, the control and the leper families were very similar if the rural and urban

groups are combined together. This proves that the selection of our control population was fairly successful in spite of serious limitations affecting the present survey.

DIET

The diet of the Cebuanos and other Visayan peoples has not been as well studied as that of the people around Manila. The Cebuano diet differs from that of the rest of the Archipelago in certain important aspects.

1. The principal grain is corn instead of rice, the latter being considered a luxury.

2. The young leaves of the kamungay tree is the commonest vegetable consumed. This vegetable is occasionally eaten by the Ilocanos, and it is practically unknown among the Tagalogs; in Cebu it is used almost daily by the poor people.

3. Since all the towns in the province are located along the seashore, the central portions of the very narrow island being mountainous and hardly habitable, fish is extensively used by practically the entire population. Among the laboring and farming classes, fish is commonly eaten in preserved form, either as "guinamus" (which is merely salted fish, often in some degree of decomposition) or as dried fish.

Corn is commonly used as a finely ground meal. In milling, the germ is removed to increase its keeping qualities. White corn is much more generally used than the yellow variety.

Corn approaches wheat in protein value, and has the same mineral deficiencies as the latter; namely, shortage of calcium, phosphorus, sodium, and chlorine, in sufficient quantities to maintain growth. It has a slightly higher caloric value than rice, and seems to be a better source of vitamin B. Beriberi is much rarer in Cebu than in Luzon.

Although corn is the basic diet of the Cebuanos, rice seems to be considered more palatable by most of them. Hospitalized lepers refuse to eat corn when rice is available. In the course of the present survey, we found that some of the poorest families sometimes bought rice and this was cooked either mixed with corn meal or alone. When this was done, however, a corresponding saving on fish and vegetables had to be made to offset the higher cost of the rice.

We have endeavored in the present survey to ascertain the frequency with which certain food items were served at the family table. When a particular item was consumed by most

of the people in the household daily or almost daily, it was considered to have been eaten "regularly." If it was served oftener than twice a week but less often than daily (say, three or four times a week), it was considered to have been eaten "frequently." If it was eaten every week or so (oftener than twice a month and less often than twice a week), the regularity was classified as "often." If an article of food was served less often than twice a month, it was considered as "seldom" eaten; if it appeared on the table only very occasionally (say, a few times a year), it was considered "very seldom" partaken of. If it had not been served as long as the informants could remember, then it was considered "never" eaten by the family.

This method of investigating the diet is naturally open to serious objections, three of which need to be mentioned here:

(*a*) This method fails to show the biologic and caloric value of the food eaten by the people under study.

(*b*) It does not indicate the consumption of the different individuals in the household.

(*c*) The data obtained are based almost entirely on information voluntarily furnished by the people in the household.

Also, with reference to the first objection, it must be mentioned that no biologic analyses have been made of many of the articles of food used in Cebu, particularly as regards the principal vegetables and the kinds of fishes eaten. It is planned to study somewhat more intensively the diet of a few representative control and leper households. The present method was used in the preliminary study merely because it was the best that could be devised with the resources available to us.

With regard to the consumption of rice and corn, the striking feature brought out in the present survey is shown in Table 27. Among households under very closely similar poor financial conditions, the leper households ate rice more commonly than the control households. It is seen in the rice column that 37.4 per cent of the control households never or very seldom ate rice, while only 10.8 per cent of the leper households ate it as seldom as that. The difference between these percentages is 26.6 ± 2.37. On the other hand, 63.6 per cent of the control families ate corn daily or almost every day, while only 8.5 per cent of the leper household ate corn as frequently. It may be concluded from the above analysis that the majority of leper households tried to eat rice as often as possible, at least more frequently than the control households. We have learned that practically every

time that rice is eaten among the poor people, less fish and vegetables are bought as rice is more expensive locally than corn. It has already been pointed out that their income is too limited to permit such luxury.

Data with respect to tubers, such as "gabi," or taro, and "ubi," etc., as well as to kamungay and other vegetables, are shown in Table 28. There is a tendency to eat tubers more frequently among the leper families than among the control households.

TABLE 28.—*Frequency of tubers, kamungay, and other vegetables on the table of control and leper families.*

Frequency at table.	Tubers.				Kamungay.			
	Control households.		Leper households.		Control households.		Leper households.	
		P. ct.		*P. ct.*		*P. ct.*		*P. ct.*
Never	13	4.2	4	11.9	6	1.9	1	0.5
Very seldom	106	34.2	20	9.4	3	1.0	1	0.5
Seldom	140	45.2	95	44.5	37	12.0	72	33.8
Often	41	13.2	91	42.8	135	43.5	132	62.0
Frequently	6	1.9	2	0.9	64	20.6	3	1.4
Regularly	4	1.3	1	0.5	65	21.0	4	1.9
Total	310	99.9	213	100.0	310	99.9	213	100.0

Frequency at table.	Other vegetables.[a]			
	Control households.		Leper households.	
		P. ct.		*P. ct.*
Never	4	1.6	6	4.0
Very seldom	1	0.4	0	0.0
Seldom	48	18.8	82	54.2
Often	109	42.5	59	39.0
Frequently	71	27.8	3	2.0
Regularly	23	9.0	1	0.7
Total	310	100.0	213	99.9

[a] No data regarding eating of "other vegetables" available for 54 control and 62 leper households.

It has been mentioned elsewhere in this report that the young leaves of the kamungay tree (*Moringa oleifera* Lam.) constitute the main leafy vegetable of the people of Cebu. Many poor families eat nothing but boiled kamungay leaves, with or without salted, dried, or fresh fish, three times a day with their corn for months at a time. So far as we are aware, the vitamin content of kamungay leaves has not been determined, although this vegetable was examined chemically long ago by Agcaoili,

who reported that it contained 7.29 per cent protein, the highest in any of the leafy vegetables studied at the time.[4] The biologic value of the protein was not determined. Agcaoili also found the cost of 1 kilo of protein in kamungay leaves much lower than in any other leafy vegetable studied by him.

Recently, this vegetable was found to be an excellent source of calcium and iron by Marañon, of the Bureau of Science. The result of his chemical analysis, heretofore unpublished, is as follows:

Leaves of kamungay, Moringa oleifera Lam.

	Per cent.
Ash:	
Fresh sample	2.84
Moisture-free sample	13.64
Phosphorus (P_2O_5):	
Fresh sample	0.24
Moisture-free sample	1.16
Calcium (CaO):	
Fresh sample	0.72
Moisture-free sample	3.47
Iron (Fe_2O_3):	
Fresh sample	0.108
Moisture-free sample	0.52

Dr. A. J. Hermano, of the Bureau of Science, has also found the leaves to be a good source of vitamins A and B_1. This vegetable is believed to stimulate the flow of human milk and it is extensively used by lactating mothers around Manila.

Referring to Table 28, it is seen that 41.6 per cent of control households ate kamungay leaves almost every day, as contrasted with only 3.3 per cent of the leper households. With regard to leafy vegetables other than kamungay, 37.8 per cent of control against 2.7 per cent of leper households, ate them as often as three or four times a week or oftener. There seems to be no doubt, then, that the majority of households in which leprosy has appeared ate less leafy vegetables than those in which the disease is unknown.

The next table (Table 29) indicates that while 39.0 per cent of the control families stated that they never or very seldom ate dried fish, only 9.9 per cent of leper families claimed to have eaten dried fish so infrequently. On the other hand, 23.6 per cent of the controls, as against 11.7 per cent of the leper households, ate dried fish at least twice a month or oftener. The

[4] Census of the Philippine Islands. Manila 3 (1918) 917–27.

large majority of the latter households admitted eating this particular food only every week or so.

Curiously, more of the control households ate guinamus, or salted fish, regularly, while raw shellfish were more frequently eaten by the leper households (Table 29).

TABLE 29.—*Frequency of dried fish, salted fish (guinamus), and raw shellfish in the diet of leper and control households.*

Frequency at table.	Dried fish.				Salted fish.			
	Control households.		Leper households.		Control households.		Leper households.	
		P. ct.		*P. ct.*		*P. ct.*		*P. ct.*
Never	10	3.2	4	1.9	11	3.5	9	4.2
Very seldom	111	35.8	17	8.0	57	18.4	25	11.7
Seldom	116	37.4	166	78.0	143	46.2	153	71.8
Often	65	21.0	25	11.7	90	29.0	21	9.9
Frequently	4	1.3	0	0.0	4	1.3	3	1.4
Regularly	4	1.3	1	0.5	5	1.6	2	0.9
Total	310	100.0	213	100.1	310	100.0	213	99.9

Frequency at table.	Raw shellfish.[a]			
	Control households.		Leper households.	
		P. ct.		*P. ct.*
Never	79	26.0	53	25.4
Very seldom	155	51.1	41	19.6
Seldom	58	19.2	106	50.7
Often	11	3.6	9	4.3
Frequently	0	0.0	0	0.0
Regularly	0	0.0	0	0.0
Total	303	99.9	209	100.0

[a] In 7 control and 4 leper homes, no data were available as to frequency of raw shellfish at the family table.

Fish is an important element in the diet of the Cebuanos. Edible fish are commonly divided in the minds of the people into those that are "malansa," or too fishy, and those that are not so. The malansa kinds include *halwan, danguit, pata, ubod indong, bunog, ugapon, barungoy, tagutuñgán, kadlitan, awá, ihó, indangan,* and some varieties of *molmol.*

Raw fish is apparently not regularly eaten in Cebu, according to the figures shown in Table 30, but it is certainly more frequently indulged in by the leper households than by the controls. On the other hand, the control families ate cooked fish, both of the malansa and the nonmalansa varieties, more regularly than the other group.

Table 31 gives some information as to the consumption of pork, beef and chicken, and milk. Very few families (less than 1 per cent except for pork which is eaten somewhat more frequently) eat meat or meat products regularly. There was no significant difference in the consumption of these items in the two groups of households. Milk was very little used by the inhabitants under study, only about 10 per cent of the families claiming that they drank milk as often as once a week, and very probably in small amounts at that.

Finally, Table 32 confirms that part of the findings of those who have investigated the diet of Filipino families which indicates that fruits and sweets are seldom eaten among our people, at least, in connection with their meals. Less than 10 per cent of the households in both groups ate fruit and "dulces" as frequently as once a week, either during or after their meals, and only 2 families out of 481 stated that they had fruit or dulce,

TABLE 30.—*Regularity of raw fish and of cooked "malansa" and "not malansa" fish in the diets of leper and non-leper households.*

Frequency at table.	Raw fish.[a]				Cooked fish, malansa.			
	Control households.		Leper households.		Control households.		Leper households.	
		P. ct.		*P. ct.*		*P. ct.*		*P. ct.*
Never	186	68.4	57	26.8	45	14.5	38	17.9
Very seldom	46	16.9	62	29.0	33	10.6	38	17.9
Seldom	33	12.1	92	43.2	98	31.6	118	55.3
Often	7	2.6	2	0.9	78	25.2	19	8.9
Frequently	0	0.0	0	0.0	34	11.0	0	0.0
Regularly	0	0.0	0	0.0	22	7.1	0	0.0
Total	272	100.0	213	99.9	310	100.0	213	100.0

Frequency at table.	Cooked fish, not malansa.[b]			
	Control households.		Leper households.	
		P. ct.		*P. ct.*
Never	0	0.0	5	2.5
Very seldom	1	0.3	6	3.1
Seldom	78	26.3	76	38.5
Often	109	36.8	106	53.8
Frequently	63	21.3	3	1.5
Regularly	45	15.2	1	0.5
Total	296	99.9	197	100.0

[a] Among 33 control households for consumption of raw fish, information was not available.

[b] For cooked fish, not malansa, 14 control households and 16 leper households had no data.

separately or both at the same time, at the table every day or almost daily. Doubtless, there is a considerable amount of indiscriminate eating of both fruits and sweets between meals, especially among the children.

TABLE 31.—*Consumption of pork, beef and chicken, and milk in the two groups of households.*

Frequency at table.	Pork.				Beef and chicken.			
	Control households.		Leper households.		Control households.		Leper households.	
		P. ct.		*P. ct.*		*P. ct.*		*P. ct.*
Never	2	0.6	5	2.4	8	2.5	1	0.3
Very seldom	97	31.3	37	17.4	115	37.1	50	23.6
Seldom	109	35.2	113	53.0	116	37.4	130	61.0
Often	91	29.4	41	19.2	68	22.0	30	14.1
Frequently	6	1.9	7	3.3	2	0.6	0	0.0
Regularly	5	1.6	10	4.7	1	0.3	2	0.9
Total	310	100.0	213	100.0	310	99.9	213	99.9

Frequency at table.	Milk.			
	Control households.		Leper households.	
		P. ct.		*P. ct.*
Never	171	55.0	109	51.2
Very seldom	51	16.5	28	13.2
Seldom	56	18.1	55	25.8
Often	21	6.8	17	8.0
Frequently	6	1.9	3	1.4
Regularly	5	1.6	1	0.3
Total	310	99.9	213	99.9

TABLE 32.—*Consumption of fruits and sweets with the meals.*

Frequency at table.[a]	Households.			
	Control.		Leper.	
		P. ct.		*P. ct.*
Never	159	58.0	84	40.6
Very seldom	42	15.3	41	19.8
Seldom	55	20.1	70	33.8
Often	15	5.5	11	5.3
Frequently	1	0.4	1	0.5
Regularly	2	0.7	0	0.0
Total	274	100.0	207	100.0

[a] No data on 36 control and 6 leper households.

TABLE 33.—*Ownership of carabaos, horses, and pigs in control and leper households.*[a]

Number of animals.	Carabaos.				Horses.			
	Control households.		Leper households.		Control households.		Leper households.	
		P. ct.		*P. ct.*		*P. ct.*		*P. ct.*
1 to 3	28	10.7	22	11.1	4	1.5	13	6.5
4+	1	0.4	0	0.0	0	0.0	1	0.5
No animal	233	89 0	177	88.9	258	98.5	185	93.0
Total	262	100.1	199	100.0	262	100.0	199	100.0

Number of animals.	Pigs.			
	Control households.		Leper households.	
		P. ct.		*P. ct.*
1 to 3	165	63.0	121	60.8
4+	20	7.6	15	7.5
No animal	77	29.4	63	31.7
Total	262	100.0	199	100.0

[a] In the three tables showing ownership of animals, no information is available in 48 control and 14 leper households.

TABLE 34.—*Ownership of goats and cows among control and leper households.*

Number of animals.	Goats.				Cows.			
	Control households.		Leper households.		Control households.		Leper households.	
		P. ct.		*P. ct.*		*P. ct.*		*P. ct.*
1 to 3	19	7.3	17	8.5	5	1.9	9	4.5
4+	3	1.1	4	2.0	0	0.0	0	0.0
No animal	240	91.5	178	89.5	257	98.1	190	95.5
Total	262	99.9	199	100.0	262	100.0	199	100.0

TABLE 35.—*Varieties of animals owned.*

Number of varieties.	Households.			
	Control.		Leper.	
		P. ct.		*P. ct.*
No animal	65	24.8	49	24.6
One kind only	161	61.4	107	53.7
Two kinds	31	11.8	34	17.1
Three or more kinds	5	1.9	9	4.5
Total	262	99.9	199	99.9

Summarizing the information obtained by the simple method that has been employed in the present report, we may say that the main differences in the diet of the control and the leper households seemed to lie in the fact that the latter tended to eat more of the more expensive rice in addition to their staple grain of corn, at the sacrifice of their vegetables and fish, especially fresh fish. The consumption of meat and meat products was about the same in the two groups. There was also noted an undoubted more-marked predisposition to the eating of raw shellfish as well as uncooked fish among the households where leprosy has appeared than among households living in the same localities and of about the same economical status, which have been respected by the disease.

OWNERSHIP OF DOMESTIC ANIMALS OF ECONOMIC VALUE

The families under study, being poor, had very few work animals. This is shown in Tables 33, 34, and 35. About 11 per cent in both control and leper groups had carabaos. The proportion of households owning horses, pigs, goats, and cows is likewise about the same in both groups. Table 33 shows that the pig is the commonest domestic animal; only about 30 per cent of both sets of households have no pigs.

Table 35 requires a brief explanation to clarify its meaning. In this table, the households are classified according to the number of varieties of domestic animals of economic value owned by each household. In other words, the figures indicate the number of kinds of animals, rather than the number of animals. When the control and the leper households are compared on this basis, no significant difference is noted.

ANALYSIS OF DATA ON THE ACTUAL CASES OF LEPROSY AND THEIR CONTACTS

POSITIVE LEPERS

There had developed in the "positive leper" households surveyed, a total of 126 cases of leprosy, of whom 82 were males and 44 were females; 15 males and 19 females (total, 34 cases) were found in Cebu, while the rest (67 males, 25 females, total, 92 cases) occurred in Opon, Mactan Island.

Tables 36, 37, and 38 give data regarding the age and sex distribution, occupation, and literacy among these positive cases. The totals appearing in these tables are not the same in the three tables because whenever data regarding the subject of the table were lacking, the cases were not included in the tabulation.

For instance, as regards age and sex distribution, complete data were available only in 117 of the total of 126 lepers; in 9 cases (4 males and 5 females) the data as to age were lacking so these do not appear in Table 36. This table shows that 55, or 47.0 per cent, of the 117 cases were under 20 years of age at the time the diagnosis was made. There were 7 children under 10 years.

TABLE 36.—*Age and sex distribution of cases of leprosy and suspects.*

Age.	Incipients.			Positives.			Paroled.			Suspects.		
	M	F	Total.	M	F	Total.	M	F	Total.	M	F	Total.
Yrs.												
1	0	0	0	0	0	0	0	0	0	0	0	0
1	0	0	0	0	0	0	0	0	0	0	0	0
2	0	0	0	0	0	0	0	0	0	0	0	0
3	0	0	0	0	0	0	0	0	0	0	0	0
4	0	0	0	0	0	0	0	0	0	0	0	0
5 to 9	5	6	11	4	3	7	0	0	0	1	1	2
10 to 14	12	9	21	11	11	22	1	1	2	0	3	3
15 to 19	14	16	30	18	8	26	4	2	6	3	0	3
20 to 29	21	13	34	28	14	42	9	2	11	0	1	1
30 to 39	3	3	6	10	1	11	6	2	8	0	0	0
40 to 49	2	2	4	1	1	2	4	2	6	0	0	0
50 to 59	0	3	3	1	0	1	0	0	0	0	0	0
60 to 69	0	0	0	5	0	5	1	0	1	0	0	0
70 to 79	0	0	0	0	1	1	0	1	1	0	0	0
80 to 89	0	0	0	0	0	0	0	0	0	0	0	0
90+	0	0	0	0	0	0	0	0	0	0	0	0
No data	0	0	0	4	5	9	0	0	0	0	0	0
Total	57	52	109	82	44	126	25	10	35	4	5	9

TABLE 37.—*Occupations of cases of leprosy.*

Occupation.	Incipients.			Positives.			Paroled.			Suspects.		
	M	F	Total.	M	F	Total.	M	F	Total.	M	F	Total.
Domestic	0	1	1	0	1	1	0	0	0	0	0	0
Employee	5	0	5	2	0	2	1	0	1	0	0	0
Farmer	7	1	8	6	0	6	3	0	3	0	0	0
Fisherman	0	0	0	6	0	6	2	0	2	0	0	0
Merchant	1	1	2	0	0	0	0	0	0	0	0	0
Ordinary laborer	7	2	9	15	3	18	11	0	11	0	0	0
Skilled laborer	8	3	11	2	3	5	0	0	0	0	0	0
Professional	0	0	0	0	0	0	0	0	0	0	0	0
Property owner	0	0	0	0	0	0	1	0	1	0	0	0
Student	14	11	25	12	12	24	1	0	1	0	4	4
Housework	0	25	25	0	7	7	0	8	8	0	0	0
No occupation	10	0	10	9	4	13	6	2	8	3	1	4
Total	52	44	96	52	30	82	25	10	35	3	5	8

TABLE 38.—*Literacy among cases of leprosy.*

Language.	Incipients.			Positives.			Paroled.			Suspects.		
	M	F	Total.	M	F	Total.	M	F	Total.	M	F	Total.
English and Spanish	0	0	0	1	2	3	0	0	0	0	0	0
English	37	25	62	22	13	35	7	0	7	0	1	1
Spanish	0	0	0	0	0	0	0	0	0	0	0	0
Visayan	4	2	6	8	3	11	6	2	8	1	0	1
Illiterate	11	13	24	20	11	31	12	8	20	3	3	6
Total	52	40	92	51	29	80	25	10	35	4	4	8

As to occupation, Table 37 indicates that of 82 individuals above 10 years old about which this item was known, 6, or 7.3 per cent, were fishermen; 6, or 7.3 per cent, were farmers; only 5, or 6 per cent, were skilled laborers; 18, or 21.8 per cent, were ordinary laborers; 24, or 29.2 per cent, were students; and 13, or 15.8 per cent, had no occupation.

The history of contact with a previous case was carefully investigated in every case of leprosy. In 16 cases, the information obtained was considered doubtful and they were excluded from the following discussion. Among the remaining 110 cases, 88, or 80 per cent, gave a definite history of contact with a leper previous to the onset of the disease. In 48, or 43.63 per cent, the leper source of the infection lived in the same household; in 23, or 20.9 per cent, the source was a relative who lived outside the household; while in 17, or 15.5 per cent, the source was a nonrelative who lived outside the household. In the last class the leper source was usually a neighbor or a playmate.

It has already been stated that only 34 positive lepers from Cebu city were included in the survey. It will also be recalled that there were 32 "positive leper" families visited. There were only two families in which more than one case appeared in the household.

On the other hand, there were 56 "leper" homes on Mactan Island surveyed and among them, there were 92 positive lepers found. Calling the first case recorded in a household the "primary" case and the subsequent ones "secondary" cases for lack of better terms, there were therefore 56 primary and 36 secondary cases. It must not be inferred from the use of these terms that the latter were necessarily derived from the former, since both may have been infected from the same source, and in the primary cases the disease simply manifested itself earlier.

At any rate, there were 44 male and 12 female "primary" cases, while 23 males and 13 females composed the "secondary" cases. As a rule, the secondary cases were somewhat younger at the time of segregation than the first cases.

It would seem from the above discussion that there exists a difference between the urban and the rural groups in that there are more multiple cases occurring among the latter. This difference is probably more apparent than real on account of the following circumstances:

1. The "positive" households surveyed in the city were visited sooner after detection of the leper and the cases as a group were more recent than the ones studied at Opon.

2. A list of all lepers whose names appear in the records as having been segregated from Opon since 1904, was prepared in 1929 and many of their homes had been located previous to the present survey. Although only recent ones are included in the present investigation, we had more definite information about the previous cases in Opon than in Cebu. In many instances, the head of the family at Opon either did not mention or actually denied the existence of previous cases in the family until the records were shown to him. It has not been possible to prepare a similar list for Cebu, and it is possible that many of the lepers reported there as "primary" will turn out to be "secondary" cases later on.

3. The population at Opon was more stable. In the city, there is apt to be more shifting about of the population.

4. The rural people were much more coöperative, and the inhabitants of Opon were better informed about the affairs of their neighbors than those in the city.

Of 1,370 persons exposed to 213 (primary) cases of leprosy (incipients, positives, and paroled combined), 57 developed the disease subsequently, which gives a rate of 4.16 per cent. It must be remembered that the subsequent cases did not necessarily develop from the first recorded case in the household.

Of 617 persons exposed to 88 positive cases, 38 developed leprosy, which is an incidence of 6.14 per cent.

The duration of the exposure has not been taken into account in the above discussions. This matter will be taken up in a subsequent report. In order to facilitate further analyses of these cases definitely diagnosed as leprosy, a graph for each household, indicating the age of each person in the household including the lepers, is being prepared. The lepers are represented by red lines and the nonleper contacts by blue lines.

By means of symbols, it is possible to show in the same chart the progress of the disease among those becoming leprous. Thus, at a glance, an idea may be had of the age and sex constitution of the contacts to a particular case, as well as the duration, course, etc., of the disease in the leper. By means of this graph, it will be possible in later years to correlate the frequency of secondary cases to the duration of exposure, the average age of the contacts, during this period, and the time that had elapsed since the leper was segregated. It should be possible in selected cases also to correlate incidence of secondary cases to type of lesion found in the leper.

The results should prove interesting in view of the fact that we are at present observing a number of "closed" cases and even "paroled ex-lepers" in newly established families.

INCIPIENT LEPERS

Coming now to the incipient or "closed" cases, there were 109 in all, from both districts, 57 being males and 52 females. The data regarding them also appear in Tables 36, 37, and 38.

There were 91 families with incipient lepers, so that there were 18 "secondary" cases.

In age distribution, 62, or 56.8 per cent, were under 20 years old. As was to be expected, they were, as a group, younger than the "positive" cases among whom 47 per cent were of corresponding age.

As regards occupation, there were no fishermen among them; 8, or 8.3 per cent, were farmers; 11, or 11.4 per cent, were skilled laborers; 9, or 9.3 per cent, were ordinary laborers; 25, or 26 per cent, were students; 10, or 10.4 per cent, of those above 10 years had no occupation, while 11 were children under 10 years of age.

It is interesting to observe that the relative number of fishermen among the lepers is less than among the control population.

There were relatively more skilled laborers among the unaffected individuals than among the positive cases and about the same proportion of students. There were fewer jobless among the former, too. It should be borne in mind that these were "closed" cases and some of them may never progress to the "positive" stage.

As to literacy, it was possible to gather data only in 92 cases above 10 years of age, of whom 24, or 26.1 per cent, were illiterates. This record compares favorably with that of 38.7 per

cent illiteracy among the positive lepers and 38.5 per cent in the control population.

In attempting to obtain the history of previous contact with a definite bacteriologically positive case of leprosy among these incipient cases, we failed to obtain reliable data in 38 instances. Among the remaining 71, 48, or 67.6 per cent, gave a history of definite contact with an undoubted "positive" leper; in only 9, or 2.7 per cent, was the leper in the household. Twenty-nine had been in contact with leper relatives outside the household and 10 gave a history of contact with a nonrelative leper also outside the household.

There was noted a much higher percentage of household infection among the positive or open cases of leprosy than among the "closed" or "incipient" cases.

PAROLED EX-LEPERS

There were 35 paroled ex-lepers in 34 homes. In 8, or 22.6 per cent, the age was below 20 years, indicating that they were older than the "positive" and "incipient" lepers. Eleven, or 31.4 per cent, were ordinary laborers and 8, or 22.6 per cent, had no jobs.

SUSPECTS

Nine cases were found to have lesions suspected of leprosy by the field workers, but due to lack of money for transportation they could not report to the dispensary for confirmation of the diagnosis. Eight were previously associated with positive lepers, while one was living with an incipient case.

SUMMARY

In December, 1932, the writers started to gather epidemiologic data on the recently discovered cases of leprosy found in the city of Cebu and its environs. The main focus of leprosy in the province had been found to be located in a roughly circular area with a radius of about 20 kilometers and with the capital at its center. Cases coming from more distant areas could not be studied due to lack of funds. However, an effort was made to study as many cases as possible of those discovered in the town of Opon, on Mactan Island, across the narrow strait from the city of Cebu, in order to include cases from a rural district in the study. The individual histories of the patients were investigated, and the economic, hygienic, and social conditions of the households in which they had been living since the probable

onset of the disease, were also studied. In this manner, it was hoped that some idea might be had of the conditions that favor the development of leprosy. It was obviously important to have controls, consisting of normal persons and households living in the same area as the lepers and the households that have been affected by leprosy.

It was decided to survey particularly the entire barrio of Baud in the San Nicolas district of the city of Cebu, as well as 5 barrios of the municipality of Opon, on Mactan Island, all of which were known to be heavily infected with leprosy. We had planned to examine every person in these barrios for signs of leprosy, and to survey all the households in them, including of course those in which the disease had developed. However, due to the reduction of personnel, following the reorganization of the Government in March, 1933, the investigation could not be continued as intensively and extensively as originally planned. After continuing for two more months, it was decided to analyze the data then available, in order to be able to replan the investigation on the basis of the reduced personnel with more adequate knowledge of actual conditions.

Up to the end of May, 1933, 95 control (498 persons) and 98 "leper" households (649 individuals) had been surveyed in the city of Cebu. By "leper" households or families are meant those households in which one or more cases of leprosy were definitely known to have developed. The individuals in such households may therefore be considered "contacts" to the disease, except those who were born after the leper had been segregated. The control households consisted of those which, in spite of the fact that they were living in an endemic area of leprosy with the "leper" households, have not been known to have been affected with the disease. These families constitute the "urban" group studied.

In the municipality of Opon, 330 "rural" households, representing 1,983 persons of both sexes and all ages, were visited. Of these households, 215 were control and 115 were "leper" families. In all, there have been surveyed, therefore, 310 control (1,817 persons) and 213 "leper" households (1,313 persons) or a total of 523 families, representing 3,130 persons. This is only about one-third of the total population of Baud and of the 5 barrios of Opon that we desire to survey.

Due to the fact that the entire population of these barrios has not been examined, it has not been possible in the present

report to determine the incidence of leprosy or its rate as to sex, by age groups, by occupations, by incomes, as to literacy, etc. Naturally, this has proven an unsurmountable handicap in the analysis of our data, and we have been forced to use less dependable methods of analysis. However, we consider the results of sufficient interest to merit publication.

Moreover, towards the middle of July, 1933, a similar epidemiologic survey was started in the municipality of Cordova, which also lies on Mactan Island, under the auspices of the Leonard Wood Memorial, by Dr. James A. Doull, with the cooperation of the personnel of the Bureau of Health. With the ample resources available for this special survey and the matured experience of Doctor Doull as an epidemiologist, there can be no doubt that the entire population of Cordova will be examined and accurate data regarding the households will be secured. It will then be possible to determine the incidence of the disease as well as to work out rates as to age, occupation, etc. The result of this survey will doubtless be an important contribution to the epidemiology of leprosy.

In the first section of the present report, the total population of the normal and the "leper" groups of households are compared as to age and sex distribution, occupation, literacy, and mortality.

In the following section, the two sets of households are compared as to type of houses, overcrowding, amount and kind of vegetation about the houses, sanitary condition, size of family, family income, diet, and ownership of domestic animals.

Finally, the last section takes up the analysis of the data regarding the lepers themselves with regard to age and sex distribution, occupation, and literacy.

CONCLUSIONS

1. Comparing the total population of the households where leprosy had developed with that of the control households as to age and sex, it was found that the mean ages of both sexes were higher in the former group than in the latter; no significant difference in the proportion of males to females was noted. There was more illiteracy among those living in leper households and the death rate was higher, although the latter was not statistically significant. There was no important difference as to occupation.

2. Comparing the control and "leper" households as to type of house and overcrowding, we have found that the latter as a

rule were housed in better homes which were not as crowded together as the controls, but that there was more overcrowding within the houses themselves.

3. With regard to amount of vegetation about the house, the "leper" houses had less vegetation about them than the controls.

4. As to sanitary conditions, the "leper" households were using, on the average, drinking water of better sanitary quality and also had safer means of sewage disposal, but were dirtier both as to surroundings and with regard to their persons than the controls. They had less cleanly habits as indicated by the more frequent presence of large and ill-smelling "pusali" under their houses and less frequency of bathing among them.

5. The leper families were larger than the controls.

6. The family income was about the same in the control and the leper households. Both groups, however, belong to the poorer or possibly the poorest classes in Cebu Province. When the population of the province is taken as a whole, the disease will be found to attack chiefly the poorest households.

7. The main differences in the diet of the control and the leper households consisted of the fact that the latter tended to eat more rice which is more expensive than corn, the local staple grain, at the expense of their vegetables and fish, specially fresh fish.

The consumption of meat and meat products was about the same in the two groups. However, these food items, except pork, seldom appear on the poor man's table in Cebu.

The "leper" households tended to eat more raw shellfish as well as uncooked fish than the controls.

8. Forty-seven per cent of the "positive" lepers were under 20 years of age when the diagnosis of leprosy was made. Among the "closed" or "incipient" cases, 57 per cent were of corresponding age at the time of diagnosis.

9. Leprosy was not associated with any particular occupation in the areas studied.

10. The proportion of illiterates among the lepers was not different from that in the general population of corresponding age and sex.

11. It was possible to obtain an authenticated history of contact with a previous case of leprosy in 80 per cent of the "positive" lepers; in 44 per cent, the source of infection had lived in the same household. Among the "closed" cases, however, the source could be traced only in 68 per cent of the cases and only in 3 per cent had the leper source lived in the same house.

EXPERIMENTS ON THE CONTROL OF THE COMMON WATER LEECH, HIRUDINARIA MANILLENSIS [1]

By ZACARIAS DE JESUS

Of the Veterinary Research Division, Bureau of Animal Industry, Manila

TWO PLATES

INTRODUCTION

The common water leech, or carabao leech, *Hirudinaria manillensis* (Lesson) (= *Hirudo boyntoni* Wharton, 1913) and in the vernacular *lintá* or *lintang kalabao*, is abundant enough in the Philippines to be of considerable economic importance. This species of medicinal leech, according to Harding and Moore (1917), occurs also in Java, Ceylon, Siam, and southern India. It is found in great numbers in ponds, pools, brooks, and irrigation ditches that are frequented by carabaos and in rice paddies that contain water throughout or during the greater part of the year.

The carabao leech is an injurious and dangerous annelid. It attacks not only carabaos that wallow in its habitat but also cattle which drink there. Human beings who work in rice paddies or fish in leech-infested places are subject to attack. People and carabaos suffer considerably from its bite. Owing to its notoriously sanguivorous habit and to its size, one mature *H. manillensis* can suck about 10 to 20 cubic centimeters of blood so that a great number of mature leeches attacking a carabao at the same time will draw a large amount of blood. It is not uncommon to see a carabao with several engorged leeches dangling from its sides and belly when it leaves a wallow. Sometimes one or more leeches stick to the muzzle or invade the nostrils of a carabao; the resulting annoyance and pain cause the animal to run aimlessly, snort violently, and rub its nose and mouth against plants or the ground. Cattle are annoyed in the same way when they drink in leech-infested

[1] Bureau of Animal Industry Technical Bulletin 4. Received for publication August 10, 1933. Read in part before the Second Philippine Science Convention, February 14, 1933.

places and possibly horses may be also. Experimentally and in nature, the carabao seems not to notice the leeches biting and sticking to its skin except when on the muzzle or around the anus. Leech bites are dreaded by most people who work in infested rice paddies, because, besides the tickling sensation and itching at the points of the bites, the victim may lose much blood and the points of bites may act as portals of entry of certain pathogenic bacteria. Harding and Moore (1927) point out that the habit of sanguivorous leeches of congregating about inflamed sores or wounds and abrasions that may be already infected, together with their proneness to pass from host to host, make them almost perfect simple mechanical carriers of bacterial infections.

Hirudinaria manillensis acts as a host reservoir of certain pathogenic microörganisms. Boynton (1913) reports that this leech can retain the virus of rinderpest alive in its body for at least twenty-five days and in a virulent condition. This investigator points out that as this leech cannot transmit the rinderpest virus through bites alone it transmits the virus to healthy animals by disgorging part of the virulent blood either on the grass or into the water. Sakharov, Rosenbach, Blumer, Hamburger, and Mitchell (cited by Bass and Johns, 1912) kept malarial plasmodia alive for several days in medicinal leeches that had been allowed to draw the blood of malarial patients. However, *H. manillensis* cannot act as a host reservoir or mechanical transmitter of the trypanosome of surra. According to Boynton (1913), the trypanosome of surra does not remain alive for any length of time in the ingested blood of *H. manillensis* and this leech cannot transmit the disease by biting. Miyao (1932) reports that the possibility of transmission of yaws by the common water leeches in the Philippines is very remote. Tubangui (1932) states that by means of interrupted-feeding experiments it was determined that this water leech is unable to transmit surra either directly or indirectly.

Blood-letting by means of medicinal leeches is a practice of the past. In the nineteenth century, according to Shipley (1927), the artificial cultivation of leeches was a profitable industry in Europe as they were in great demand in European hospitals. Nachtrieb (1912) states that in those days one American leech farm sold as many as one thousand or more leeches a day. While medicinal leeches are still sold at some drug stores even in Manila, the medical profession condemns the employment of these annelids in phlebotomy. The practice,

however, is still carried on in the Philippines by the *mediquillos* or quacks, but the medicinal leech, like *Hirudinaria manillensis*, has outlived its usefulness and has become but an injurious pest which deserves extermination.

The object of the study here reported was to find some practical methods of controlling or possibly exterminating, at least in certain places, the common water leech.

MATERIALS AND METHODS

With the help of Drs. R. Q. Javier, N. S. Sevilla, M. C. Villanueva, and several students of the College of Veterinary Science, many specimens of the common water leech, *Hirudinaria manillensis*, were collected from rice paddies, brooks, and pools in Lumbang, Laguna, and in Tupas, Nueva Vizcaya, in 1930; in Papaya, Nueva Ecija, in 1931; in Pila, Santa Rosa, and Bay, Laguna, and in San Narciso, Zambales, in 1932. The leeches were kept in large bottles and jars half-filled with water, which was changed every two or three days.

Only cheap and easily available materials were used in the different killing experiments. Solutions of copper sulphate of commercial quality were prepared, varying in proportion from 1: 1,000 to 1: 1,000,000. Crude common salt (sodium chloride), prepared in the Philippines by evaporating sea water in the open during the dry season, was used in one experiment in 1 to 5 per cent solutions. Solutions of commercial chemical fertilizers, such as, ammonium sulphate, superphosphate, potassium sulphate, calcium nitrate, potassium chloride, Leunaphos IG, Corona Special, ammonium phosphate, and Double Crop No. 1, were tested for their killing power on the water leech. In eighteen flasks 1 and 2 per cent solutions were prepared in 400-cc quantities.

Infusions of certain parts of plants that are known or said to be poisonous to fish and are easily obtainable in the Islands were prepared and used in three experiments. Fresh tubers of *namí* (*Dioscorea hispida* Dennst.) were chopped into small thin chips, and infusions were made varying in proportion from 1: 100 to 1: 1,000. The fresh roots of *tuble* (Vis.) or *tibanglan* (Tag.) [*Derris elliptica* (Roxb.) Benth.] were chopped into small chips, and after drying in the room for five days, were coarsely ground in a small grinding machine. Infusions, varying in proportion from 1: 100 to 1: 1,000, were prepared from these ground roots. Both the namí and this species of *Derris*

grow wild and in abundance in thickets and margins of forests in the Philippines. Dried tobacco (Isabela variety) midribs were cut into pieces about 2 inches long and infusions of these, varying in proportion from 1:100 to 1:1,000, were used in one experiment. Truck loads of tobacco midribs are burned in the Manila Crematory almost every day.

In all cases, the solutions and infusions were placed in 500-cc Erlenmeyer flasks and in 2,000-cc, tall, cylindrical museum jars. These were covered with two layers of strong bobbinet. Four leeches of different sizes, two large and two small, were used in each trial in the different killing experiments. Observations were made every hour when the leeches were exposed to strong solutions and infusions and every three to six hours when exposed to weaker ones.

The commonest fishes in the rice paddies are the murrel, or *dalag* (*Ophicephalus striatus* Bloch); the catfish, or *hitó* [*Clarias batrachus* (Bloch)], and the climbing perch, or *tinikan* [*Anabas testudineus* (Bloch)]. Several of these fishes were caught in rice paddies and in Laguna de Bay and kept in a laboratory aquarium or in an experimental pool.

The experimental pool (Plate 2, fig. 2) was made by digging an extensive pit, 3.7 meters long, 2.6 meters wide, and 0.6 meter deep. Water was constantly supplied the pool from a faucet, which was kept running at a force that kept the depth of the water between 0.3 and 0.45 meter. The pool was provided with a 4-inch drain pipe, the proximal end of which was covered with a piece of fine wire netting to prevent the leeches from escaping with the outflowing water. The water in this pool could be siphoned off by means of a long rubber hose. To prevent the leeches from escaping with the water through the hose, the proximal end of the tube was covered with two layers of gauze. To make it a balanced aquarium and like a rice paddy or a natural pool, rice of lowland variety, taro or *gabi*, and *cangcong* [*Ipomoea reptans* (Linn.) Poir.] were planted in the corners and center. Fifty-two snails of different sizes, collected from Molawin Creek, were let loose in the pool.

The domesticated ducks used in the hand-feeding and natural-feeding experiments are zoölogically known as *Anas boschas* Linn. and vernacularly known as *itek* or *bebe* (Plate 2, fig. 1). This species of duck is extensively raised along the shores of Laguna de Bay and along the banks of Pasig River, as well as on the banks of ponds and rivers in other parts of the Philippines.

For convenience, the leeches were divided arbitrarily into two groups; namely, young leeches (Plate 1, fig. 2), which measured from 3.71 to 6.25 cm long, and mature leeches (Plate 1, fig. 1), which measured from 7.62 to 11.43 cm long.

EXPERIMENTS AND RESULTS

TO DETERMINE THE RELATIVE KILLING POWER ON HIRUDINARIA MANILLENSIS OF SOLUTIONS OF COPPER SULPHATE

Experiment 1.—The solutions of copper sulphate (commercial) in 500-cc quantities were placed in three flasks and three jars. Four leeches of different sizes—that is, two young ones and two mature ones—were put in each solution. The leeches were examined every hour, every six hours, or every day for one week, depending upon the strength of the solution. To determine whether or not the leeches were living, they were touched with one end of a stirring rod; and, in case of doubt, they were removed from the solution and were placed in tap water in a large beaker and observed for vitality. This experiment was repeated and the same procedure was followed. The results, which were the average of those obtained from two trials are shown in Table 1.

TABLE 1.—*Showing the relative killing power on Hirudinaria manillensis of various solutions of copper sulphate.*

Solution.	Results of exposure of—	
	Young leeches.	Mature leeches.
1:1,000	Died in 1 hour	Died in 2 to 2.5 hours.
1:10,000	Died in 2 to 3 hours	Died in 5 to 7 hours.
1:50,000	Died in 18 to 24 hours	Died in 48 to 75 hours.
1:100,000	Died in 48 to 67 hours	Not affected.
1:500,000	Died in 10 days	Do.
1:1,000,000	Not affected	Do.

TO DETERMINE THE RELATIVE KILLING POWER ON THE CARABAO LEECH OF SOLUTIONS OF CRUDE COMMON SALT

Experiment 2.—Solutions of crude common salt were prepared in five flasks, in 300-cc quantities. As in experiment 1 four leeches of different sizes were placed in each solution. The leeches in the higher solutions were examined for their behavior and vitality every two or five minutes, and those in 1 and 2 per cent solutions, every six hours. This experiment was repeated; the average of the results from two trials are shown in Table 2.

TABLE 2.—*Showing the relative killing power on H. manillensis of solutions of crude common salt.*

Solution.	Results of exposure of—	
	Young leeches.	Mature leeches.
Per cent.		
1	Not affected	Not affected.
2	Died in 12 to 24 hours	Died in 48 to 75 hours.
3	Died in 22 minutes to 2.5 hours	Died in 3 to 7 hours.
4	Died in 17 to 25 minutes	Died in 1 to 2 hours.
5	Died in 10 to 15 minutes	Died in 30 to 70 minutes.

DETERMINATION AS TO WHETHER OR NOT SOLUTIONS OF COMMERCIAL CHEMICAL FERTILIZERS WILL KILL THE CARABAO LEECH

Experiment 3.—Four leeches of different sizes, as in experiment 1, were put in each solution. Observation for vitality was made every six or twelve hours. All the leeches in the 1 and 2 per cent solutions remained alive even after a continuous exposure of one month.

DETERMINATION AS TO WHETHER OR NOT INFUSIONS OF NAMÍ WILL KILL THE CARABAO LEECH

Experiment 4.—Infusions of namí tubers in 500-cc quantities were placed in five museum jars, and four leeches were placed in each of the jars. After an exposure of one week it was found that even the 1:100 infusion did not affect the leech at all.

TO DETERMINE THE KILLING POWER ON THE CARABAO LEECH OF INFUSIONS OF TOBACCO MIDRIBS

Experiment 5.—The same technic as in experiment 4 was followed. Observations were made every five minutes in case of the stronger infusions and every one or six hours in case of the weaker ones. This procedure was repeated under similar conditions. The averages of the results are shown in Table 3.

TABLE 3.—*Showing the relative killing power on H. manillensis of the infusions of tobacco midribs.*

Infusion.	Results of exposure of—	
	Young leeches.	Mature leeches.
1:100	Died in 15 to 17 minutes	Died in 37 to 42 minutes.
1:200	Died in 1 hour to 3 hours 22 minutes	Died in 11.5 to 24 hours.
1:400	Died in 12 to 24 hours	Died in 28 to 72 hours.
1:600	Died in 5 days	Died in 7 days.
1:800	Not affected	Not affected.
1:1,000	do	Do.

TO DETERMINE THE RELATIVE KILLING POWER ON THE CARABAO LEECH OF INFUSIONS OF DERRIS ELLIPTICA ROOTS

Experiment 6.—The same technic as in experiment 4 was followed, except that the derris roots were coarsely ground before being used. This experiment was also repeated under similar conditions; the averages of the results are shown in Table 4.

TABLE 4.—*Showing the relative killing power on H. manillensis of infusions of Derris elliptica.*

Infusion.	Results of exposure of—	
	Young leeches.	Mature leeches.
1:100	Died in 1 to 2.5 hours	Died in 6.5 to 17 hours.
1:200	Died in 3 to 3.5 hours	Died in 10 to 21 hours.
1:500	Died in 7 to 13 hours	Died in 21 to 33 hours.
1:700	Died in 14 to 29 hours	Died in 65 to 72 hours.
1:1,000	Died in 24 to 48 hours	Died in 54 to 75 hours.

DETERMINATION AS TO WHETHER OR NOT TEMPORARY DRAINING OR DRYING AN INFESTED PLACE WILL KILL THE CARABAO LEECH

Experiment 7.—Three empty kerosene cans were thoroughly cleaned to remove traces of kerosene. About fifty holes, 3 mm in diameter, were punched in the bottom of each can for drainage. The cans were filled with a mixture of one part of fresh carabao manure and fifty parts of loose soil. With a liberal amount of water and by thoroughly stirring this mixture was given the consistency of rice-paddy mud.

August 23, 1932. Three leeches, varying in length from 6.25 to 11.43 cm, were allowed to bury themselves in each of the three mud preparations. The leeches readily wiggled their way into the mud. Every sunny day, these leech cultures were placed in the sun from 7 o'clock in the morning to 5.30 in the afternoon; but on rainy days and every night, they were kept under the eaves of a house. Every night the cans were covered tightly with fine wire netting to prevent the leeches from escaping.

September 23, 1932. One month later, one leech culture was examined for living leeches. The upper part of the mud was dry and hard and was traversed by large deep cracks. No leech could be seen in the cracks. When the surface of the mud was liberally covered with water, a large vigorous leech appeared in the water ten minutes later. A thorough examination of the mud revealed that the leeches probably remained in the lower

layer of the drying mud where they bored tunnels communicating with the cracks in the upper layer. The walls of the lower end of one tunnel were found covered with a thin layer of thick mucoid substance. No traces, however, of the other two leeches could be found.

October 23, 1932. Two months later, a second leech culture was examined for living leeches. Three tunnels were found in the dried mud, but neither living leeches nor traces of their bodies could be found. It is highly probable that the leeches in this culture escaped, because on October 10, 1932, a large fully engorged leech was found in a small shallow mud hole just 3.5 meters from the leech cultures. No leech had ever been found in this place before, and, being well drained, it was not likely that leeches could breed in this place. This particular place was frequented every night by five experimental carabaos so the leeches in the culture must have been attracted by their odor and escaped from the culture. On testing the efficiency of the wire-netting cover of this can, it was found that a leech could easily escape by passing between the lip of the can and the wire netting.

November 23, 1932. Three months later, the last leech culture was examined for living leeches. Thirty minutes after the dried mud was flooded, no leech had appeared in the water. The dried mud was then broken into small pieces and particular care was taken to look for tunnels made by the leeches. In the lower ends of two tunnels two living leeches were found covered with thick mucoid substance, and when they were placed in water, they showed their usual vigor.

Experiment 8.—September 5, 1932. Four leeches, two mature and two young ones, were placed in a cylindrical museum jar, 14 inches high and 4 inches in diameter, in which there was a mixture of one part of fresh carabao manure and fifty parts of mud. The jar was tightly covered with a double layer of bobbinet and was placed on a table near a window where every afternoon it was exposed to the sun for about three hours. The leeches could be observed moving in their tunnels close to the wall and near the bottom of the jar.

November 3, 1932. Examination of this leech culture was made by emptying the contents of the jar on a cement floor where the mud was broken into small pieces. Three living, vigorous leeches, one young and two mature ones, were found at the bottom of the jar. The whole contents, including the leeches, were returned to the jar for further observation.

December 20, 1932. On again examining the culture, two living, vigorous leeches, one young and one mature, were found at the bottom of the jar. Many tiny, young earthworms were found also in the mud. The whole contents, including the leeches, were returned to the jar for further observation.

January 9, 1933. Over four months from the time it was prepared, this leech culture was examined for the third time. Two living leeches were found feeding on young earthworms; they appeared more plump and vigorous than they had been before.

DETERMINATION AS TO WHETHER OR NOT THE FISHES COMMONLY FOUND IN RICE PADDIES FEED ON THE CARABAO LEECH

Experiment 9.—March 27, 1932. Four murrels, or dalag, from 28 to 36 cm long, were placed in a large laboratory aquarium, 50 by 30 by 30 cm, together with three young leeches from 3.71 to 5 cm long. Five days later the number of leeches remained the same and the four fishes were still vigorous.

August 10, 1932. Two large dalag, one 35.5 cm long and the other 43 cm long, were placed in the experimental pool together with six leeches from 3.71 to 8.89 cm long. On the second day, one large leech was actually seen chasing and attacking a murrel, but no murrel was observed attacking a leech. Three days later, the pool was drained in order to catch the fish and to count the leeches. One murrel was found bleeding from leech bites over almost the whole body; the other had four bites on different parts of the body. All the original six leeches were recovered.

Experiment 10.—April 3, 1932. Two catfishes, or hitó, one 23 cm long and the other 30 cm long, were put in a large laboratory aquarium together with four small leeches, each measuring 3.71 cm long. For eight days it was observed that the number of leeches remained the same.

August 15, 1932. Four catfishes, each 20.5 cm long, were placed in the experimental pool together with four leeches, three of which measured from 3.71 to 6.25 cm long and one 10 cm long. Three days later, the pool was drained. The catfishes were found unhurt, and the four original leeches were recovered.

Experiment 11.—April 20, 1932. Two climbing perches, or tinikan, each measuring 12.5 cm long, and three small leeches, each measuring 3 cm long, were put together in a large laboratory aquarium. After thirty-six hours of observation it was found that neither the fish had attacked the leeches nor the leeches attacked the fish.

TO DETERMINE WHETHER OR NOT ANAS BOSCHAS, OR ITEK, FEEDS ON THE CARABAO LEECH

Experiment 12.—May 12, 1931. Three leeches, from 5 to 10 cm long, were placed in a large white enameled wash basin containing about a liter of water. The basin was then placed near a flock of eleven ducks. No sooner had the ducks seen the leeches than they fought for them. The smaller leeches were swallowed very easily with a little water. However, the duck that swallowed the largest leech, 10 cm long, twisted its neck several times and drank water at short intervals. Two hours later, this duck was seen going about in search of food as usual.

Experiment 13.—April 1, 1932. The walls and the bottom of a cement drinking trough, 60 by 60 by 30 cm, were smeared thickly with dark mud. Water was allowed to flow into the trough to a depth of 15 cm, and two leeches, one 7.5 cm long and the other 5 cm long, were placed in the water. A female duck was put in the trough and it was then covered with chicken wire. It was observed that before the duck was put into the trough the leeches remained quiet and clung to the sides. Upon sensing the presence of the duck the leeches swam about as if to attack the intruder, but they were swallowed by the duck.

This experiment was repeated twice (April 2 and 8, 1932). In each case, however, two ducks and larger leeches were used. When the ducks' bodies were examined after the disappearance of the leeches from the water, neither leech nor leech bites could be found. The ducks were placed in separate cages for observation for one week during which time the birds remained normal.

Experiment 14.—April 3, 1932. A female duck was hand fed with two living leeches, each measuring 6.25 cm long. The duck was then put in a cage for observation. Twelve hours later, the entire alimentary canal from the mouth to the anus was slit open; and the mucous membrane, as well as the contents, was examined. Neither pieces of the bodies of the leeches nor points of bites could be found.

Experiment 15.—April 4, 1932. A second duck, a male, was hand fed with three living leeches, measuring from 6.25 to 8.89 cm long. Soon after swallowing the leeches, the duck twisted its neck a couple of times and then drank some water. No symptom of trouble was observed thereafter, and the bird was soon feeding as usual. Six hours later the duck was killed, and the entire alimentary tract was slit open. The same results as in experiment 14 were observed.

Experiment 16.—April 12, 1932. A third duck, a female, was hand fed with two leeches from 6.25 to 7.62 cm long. Three hours later, the duck was killed and the alimentary tract was examined. The same results as in experiments 14 and 15 were found, except that one bleeding leech bite was observed at the base of the tongue.

Experiment 17.—April 18, 1932. A fourth duck, a female, was hand fed with three living leeches each measuring 8.89 cm long. One and one-half hours later, the duck was killed and examined as in the preceding experiments. No leech bite was observed. Two dead leeches were found in the crop already mixed with the feed, and one leech was found partially ground in the gizzard. It was noted that there were twenty-four pebbles in the gizzard of one duck ranging in size from 2 by 3 by 3 to 4 by 5 by 14 mm. For the sake of clarity, the results in experiments 14, 15, 16, and 17 are shown in Table 5.

TABLE 5.—*Showing the results of feeding H. manillensis to Anas boschas.*

Date.	Duck.	Leeches fed.	Length of leeches.	Time interval.	Leech bite.	Fate of the leeches.
			Inches.	*Hrs.*		
April 3, 1932	Duck, female.....	2	2.5	12	.0	Digested.
April 4, 1932	Duck 2, male.....	3	2.5–3.5	6	.0	Do.
April 12, 1932	Duck 3, female....	2	7.5–3.0	3	1	Do.
April 18, 1932	Duck 4, female....	3	3.5	1.5	.0	Two dead in crop, one ground in gizzard.

EXTERMINATION OF LEECHES IN AN INFESTED POOL BY MEANS OF ANAS BOSCHAS

Experiment 18.—August 2, 1932. Seven leeches, varying in length from 5 to 7.62 cm, were let loose in the experimental pool. One hour later, three mature ducks, one male and two females, were allowed to swim in the leech-infested pool for eight hours. Then the water of the pool was siphoned off in order to facilitate the search for the leeches. Not a single leech was found after a careful search in the pool.

Experiment 19.—August 3, 1932. The pool was again filled with water and seven leeches, varying in length from 6.25 to 8.89 cm, were let loose in it. The three ducks used in experiment 18 were allowed to swim in the pool for three hours. The next day the water in the pool was drained. After a thorough search, two leeches, one large and one small, were found caught

in the flap of a piece of wire netting used for covering the proximal end of the drain pipe. The ducks, evidently, had not seen these leeches, and, even if they had, they probably could not have caught them.

Experiment 20.—August 6, 1932. In order to determine whether or not the ducks actually fed on leeches in the infested pool much closer observation was necessary. The experimental pool was again filled with water, and six leeches, varying in length from 6.25 to 11.43 cm, were let loose in it. When the writer dipped his hand in the water of the pool, the leeches swam toward it and attempted to bite it. The three ducks used in the preceding experiments were again allowed to swim in the pool. After five minutes, four of the leeches were seen swimming excitedly around the ducks in their attempt to attack them. On seeing the leeches, the ducks also became excited and began to charge. The smaller leeches were swallowed at once with a little water. The largest leech, measuring 11.43 cm long and about 1.5 cm wide, was caught by one duck. For five minutes, one half of the body of the leech could be seen dangling from the duck's bill. Failing to swallow this large leech, the duck went to the shallowest part of the pool and freed the leech, but instantly caught it again. After many attempts to swallow it, lasting for seven minutes, the duck managed to get it down with the help of a little mud and water. Nothing happened to this particular duck thereafter. Five hours later, the pool was drained and a search for leeches was made, but not one was found.

DISCUSSION OF RESULTS

Certain dilute solutions of copper sulphate (commercial) can be used to advantage in killing *Hirudinaria manillensis*. As shown in Table 1, a 1 : 50,000 solution killed both the young and mature leeches in from eighteen to seventy-five hours, while a 1 : 100,000 solution killed the young leeches in forty-eight to fifty-seven hours, but not the mature ones, even after a prolonged exposure. A solution of higher concentration than 1 : 50,000 is highly detrimental to the life of this leech. When exposed to a 1 : 10,000 solution, the leech swam excitedly and disgorged most of its stomach contents. About thirty minutes later, the entire body excreted a thick mucoid substance; and about one hour later, both the anterior and posterior suckers lost their power of attachment. The suckers and the genital cones became swollen and hæmorrhagic, the body contracted, then relaxed, and the

annelid died in from two to seven hours. Copper sulphate is an astringent in dilute solutions, caustic in concentrated ones (Milks, 1930). On the basis of the physiological reactions manifested by the annelid, it seems that the 1 : 10,000 copper sulphate solution is both irritating and poisonous to the leech.

A practical method of applying the copper sulphate is by wrapping the drug in a piece of cloth or gunny sack and dragging the bundle over the infested place. Another method is by broadcasting a sufficient amount of powdered copper sulphate on the water so that when the carabaos come to wallow the water will be stirred and the drug uniformly distributed. However, Moore (1923) points out that copper sulphate cannot be used successfully to kill the leeches in the lake, because the toxicity of the drug for the leeches is diminished with the increase in the quantity of minute algæ present.

Table 2 shows that a 2 per cent solution of crude common salt killed both the young and mature leeches in from twelve to seventy-five hours, and a 3 per cent solution killed them in from twenty-two minutes to seven hours. The physiological reactions shown by the leech were apparently the same as those when it was exposed to solutions of copper sulphate. According to Milks (1930), a strong solution of sodium chloride abstracts water from the cells. It may be that, in this case, the leech died through cellular dehydration if not from direct poisoning. As at these concentrations a large amount of common salt is necessary even for a medium-sized carabao wallow, it is not economical to use it as the copper sulphate may be used at much less cost.

If dilute solutions of certain commercial chemical fertilizers had deleterious effects on the carabao leech, the employment of these chemicals would have served a dual purpose in leech-infested rice paddies; but the fact that even a 2 per cent solution cannot kill the young leeches after a long exposure puts these chemicals out of the question.

It is said that when the chopped up fresh roots of namí (*Dioscorea hispida*) are soaked in water in the river in order to remove its poisonous principle, the fishes in that river are killed. The results of the present experiments, however, showed that even a 1 : 100 infusion did not affect the leeches.

Strong tobacco infusions are positively inimical to the life of the carabao leech. A 1 : 600 infusion of tobacco midribs, as shown in Table 3, killed both the young and mature leeches after an exposure of from five to seven days, but a 1 : 100 in-

fusion killed them in from fifteen to forty-two minutes. If tobacco midribs, which are otherwise waste, are available in large quantities, as in Manila, they may be utilized by dumping them into leech-infested places.

As shown in Table 4, infusions of the roots of tibanglan (*Derris elliptica*) are poisonous to the carabao leech, which succumbs to a 1 : 100 infusion in from one to seventeen hours and to a 1 : 1,000 infusion in from twenty-four to seventy-five hours. The active principle in derris, according to Tattersfield and Roach (1922), is nitrogenous-free, nonglucosidal, nonalkaloid, white crystalline derivative and resin to which they gave the name "tubatoxin," and which they claimed to be identical with "tubain" of Wray (1892) and with "derrid" of Sillevoldt (1899). Wray (1892) claimed that the active principle is lost upon drying. But the writer used the air-dried roots in the preparation of the infusions which killed the leeches. The active principle of derris, according to De Ong and White (1924), on the basis of their experiments with insects, is both a stomach and a "respiratory" or tracheal poison. A practical method of applying derris in killing leeches is by scattering a sufficient amount of ground roots in the infested water, thereby dissolving the active principle. Another method is by pounding the roots and then dragging them in the infested water.

Hirudinaria manillensis can survive in the bottom of the cracks of dried mud for over three months and in the bottom of their tunnels in constantly wet mud for over five months without showing any change in their vitality. While in this apparently unfavorable environment, they feed on earthworms or other animals therein, so that instead of dying or being in a dying condition their body growth continues unimpaired. This explains why farmers failed in their attempts to control or to exterminate the carabao leech in their rice paddies by draining the fields during the hot season and then burning the rice straw and grass thereon.

The commonest fishes in the rice paddies and pools are the murrel, or dalag; the catfish, or hitó; and the climbing perch, or tinikan. Repeated experiments showed that these fishes do not feed even on very young carabao leeches. On the contrary, the carabao leech attacks the murrel and feeds on its blood. Masterman (1908) reports that in some parts of Palestine the water at the source was kept free from leeches (*Limnatis nilotica*) by means of a kind of carp (*Capoeta fratercula*). If the murrel

fed on *H. manillensis*, it is probable that this annelid would have been exterminated long ago in the Philippines owing to the presence of this fish in places where the carabao leech abounds.

The itek (*Anas boschas*) frequents water, not only because of its mating habit but also for the purpose of searching for food. Repeated experiments showed conclusively that this duck relishes *H. manillensis*. The size and shape of its bill as well as the grinding capacity of its gizzard are well adapted for leech feeding. The pebbles found in the gizzard of this duck are, on the average, very much larger than those found in the gizzard of a domestic hen. As a rule the leeches are swallowed alive, and are killed either in the crop or in the gizzard, as shown in Table 5. One and one-half hours after being swallowed a medium-sized leech is already ground in the gizzard of a hungry duck, and it is digested three hours later. In view of the peculiar feeding habit of the duck of swallowing its food with much water, small leeches, when swallowed, seem to swim their way through the œsophagus into the crop of the bird. Large leeches, however, are swallowed only with difficulty. In spite of this, the duck does not give up until even the largest is swallowed.

Owing to the fact that *H. manillensis*, especially the mature one, is attracted by the hot-blooded animals, the carabao leech is attracted by the duck just as much as it is attracted by the carabao. The leeches, upon sensing the presence of the ducks in their habitat, swim towards the birds to attack the intruders but are themselves swallowed by the ducks. Repeated trials have shown that the itek (*Anas boschas*) is a very efficient agent in getting rid of the leeches in an experimentally infested pool.

SUMMARY AND CONCLUSION

1. Since the 1 : 50,000 solution is the weakest solution of copper sulphate found to be deleterious to both young and mature *Hirudinaria manillensis*, a solution of approximately this concentration may be employed in the destruction of leeches in pools and rice paddies by either broadcasting a sufficient amount of the powdered drug on the water or by dragging a bundle containing copper sulphate through the infested water.

2. Commercial chemical fertilizers, at least those mentioned in this paper, are not harmful to the carabao leech even at a 2 per cent concentration.

3. If the infested place is very limited in area, as a carabao wallow or a pool, a sufficient amount of crude common salt may

be broadcast on the premises to make, approximately, a 2 or 3 per cent solution.

4. Since a 1 : 400 infusion of tobacco midribs killed the carabao leech in from twelve to twenty-four hours, the midribs, which are otherwise waste, can be utilized by scattering them in infested pools to kill the leeches.

5. As a 1 : 100 infusion of tobacco and a 5 per cent solution of crude common salt are both highly inimical to the life of *H. manillensis*, killing it in from ten to forty-two minutes, these preparations should be very useful in dislodging the leeches from any part of a body, particularly from the inside of the nostrils of a carabao.

6. *Derris elliptica* infusion is markedly poisonous to the carabao leech as it is to fishes, as shown by the fact that a 1 : 1,000 infusion can kill this leech in from twenty-four to seventy-five hours; and in view of the availability of this plant, its use in the control of leeches should be practical and economical.

7. *Hirudinaria manillensis* can survive in the bottom of dried mud for over three months and in constantly wet mud for over five months, which periods are longer than the time covered by the hot season in the Philippines.

8. The experiments showed that the commonest fishes in the rice paddies and pools, such as the murrel, the catfish, and the climbing perch, do not prey upon even the smallest carabao leech.

9. The itek, or bebe (*Anas boschas* Linn.), is anatomically and physiologically adapted for feeding on leeches, and repeated experiments conclusively showed that this duck relishes *H. manillensis*.

10. As the itek relishes the carabao leech and diligently searched for it in infested pools, raising this duck in leech-infested places, which are otherwise neglected, will help in the control or extermination of leeches.

ACKNOWLEDGMENT

The writer is indebted to the College of Veterinary Science, University of the Philippines, which afforded him all the facilities to conduct the experiments in this investigation while still in Los Baños, Laguna; to Mr. Felix Erasga for kindly supplying him with any number of ducks free of charge; and to Mr. Honorio N. Reyes for his interest in collecting a great number of leeches and several species of fresh-water fishes used in the experiments.

LITERATURE CITED

BASS, C. C., and FOSTER M. JOHNS. The cultivation of malarial plasmodia (Plasmodium vivax and Plasmodium falciparum) in vitro. Journ. Exp. Med. 16 (1912) 567–579.

BOYNTON, WILLIAM HUTCHINS. Duration of the effectiveness of virulent rinderpest blood in the water leech, Hirudo boyntoni Wharton. Philip. Journ. Sci. § B 8 (1913) 509–521.

DE ONG, E. R., and L. T. W. WHITE. Further studies of Derris as an insecticide. Journ. Econ. Entomol. 17 (1924) 499–501.

HARDING, W. A., and J. PERCY MOORE. Hirudinea. The fauna of British India including Ceylon and Burma. Edited by Sir Arthur E. Shipley. London: Taylor and Francis (1927) xxxii + 302 pp., 63 figs., and 9 pls.

MASTERMAN, E. W. G. Hirudinea as human parasite in Palestine. Parasit. 1 (1908) 182–185.

MILKS, HOWARD JAY. Veterinary Pharmacology Materia Medica and Therapeutics with a chapter of biological therapeutics by Adolph Eichhorn. 2d ed. Alexander Eger. Chicago (1930) xi + 539 pp., 32 figs.

MIYAO, ISAO. Experimental inquiry into the possibility of transmission of yaws by leeches. Philip. Journ. Sci. 47 (1932) 466–467.

MOORE, J. PERCY. The control of blood-sucking leeches, with an account of the leeches of Palisade Interstate Park. Roosevelt Wild Life Bull. 2 (1923) 9–54.

NACHTRIEB, HENDRY F. General account of the habits and structure of leeches. The leeches of Minnesota. Geological and natural history survey of Minnesota. Zoölogical series No. 5 (1912) 3–21.

SHIPLEY, Sir ARTHUR E. The fauna of British India including Ceylon and Burma. London: Taylor and Francis (1927) xxxii + 302 pp., 63 figs., and 9 pls.

SILLEVOLDT, H. E. TH. VAN. Ueber das Derrid und Pachyrhizid, ein Beitrag Zur Kenntnis der indischen Fischgifte. Arch. Pharm. Bd. 237 (1899) 595–616. Cited in Journ. Agr. Res. 17 (1919) 177–200.

TATTERSFIELD, F., and W. A. ROACH. The chemical properties of D. elliptica (tuba root). Cited in Annals of Applied Biology 10 (1922) 1–17.

TUBANGUI, MARCOS A. Observations on the possible transmission of surra by the land leech, Haemadipsa zeylanica. Philip. Journ. Sci. 48 (1932) 115–127.

WRAY, LEONARD, Jr. On the Malayan fish poison called aker tuba, Derris elliptica. Pharm. Journ. and Transactions 52 (1892) 61–162.

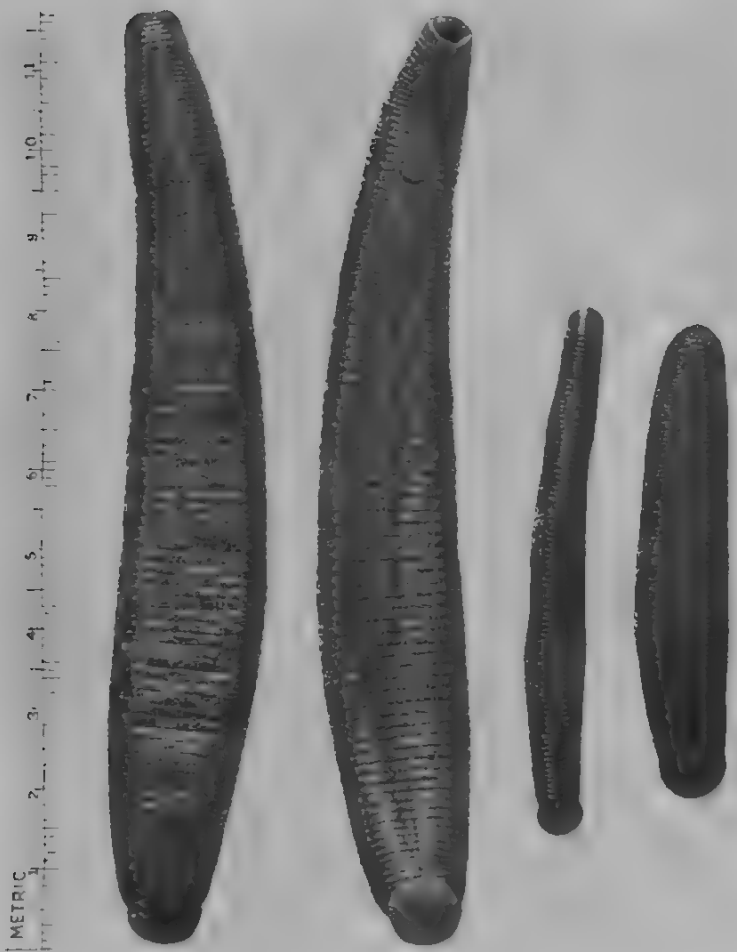
ILLUSTRATIONS

PLATE 1

FIG. 1. *Hirudinaria manillensis*, mature, dorsal and ventral views, × 1.
2. *Hirudinaria manillensis*, young, dorsal view, × 1.

PLATE 2

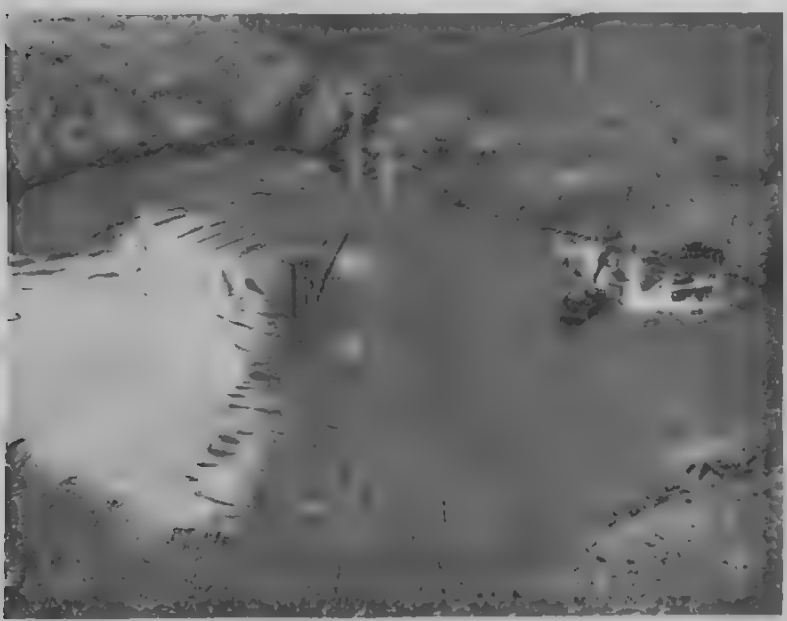
FIG. 1. *Anas boschas*, male and female, × 1/13.
2. Experimental pool with three ducks swimming in its water.



1 2

PLATE 1.

1



2

PLATE 2.

PLANT-DISEASE PROBLEMS CONFRONTING TRUCK FARMERS IN TRINIDAD VALLEY AND THE VICINITY OF BAGUIO, MOUNTAIN PROVINCE, PHILIPPINE ISLANDS [1]

By T. G. FAJARDO [2]

Plant Pathologist, Phytological Research Division, Bureau of Plant Industry, Manila

TWENTY-FIVE PLATES

Because of their semitemperate climate, Trinidad Valley and the environs of Baguio, Mountain Province, constitute one of the few regions in the Philippines where semitemperate crops can be grown successfully, and where truck gardening is extensively practiced. Fresh vegetables raised in this region find a ready market in Baguio, Manila, and nearby provinces, and as a result of this and the favorable climate, the acreage under cultivation for vegetables has greatly increased in the past ten to fifteen years, so that the yearly income from vegetables alone is conservatively estimated at several thousand pesos.

The farmers at present, in order to meet the demand for fresh vegetables, grow them extensively in succession or in short rotation throughout the season, growing plants of the same or closely allied families on the same piece of ground. Furthermore, instead of raising their own seeds, the vegetable growers find it more practical and profitable to buy them from other countries. With such farming practices, and the rather long, mild, moist season, the increase in the severity and distribution of indigenous diseases and the introduction of foreign parasites which can adapt themselves to these regions are inevitable. The farmers of this locality, therefore, are confronted not only with various soil-deficiency problems, but also with numerous plant diseases and pests which seasonally or annually destroy all or part of their crops.

[1] This work was done while the writer was connected with the Bureau of Science.

[2] Formerly plant pathologist, Bureau of Science, Manila.

Therefore, a survey of these plant diseases seems of considerable interest.[3] This paper principally aims to report and enumerate the occurrence of the important and common diseases of various garden crops grown in Trinidad Valley and the environs of Baguio, Mountain Province, with the hope that a detailed investigation may be conducted in the future on the etiology, epidemiology, and control of each of the maladies mentioned herein. For the purpose of the present studies, the host plants are divided into groups and their diseases are listed and briefly described in the order of their importance.

DISEASES OF CRUCIFEROUS CROP PLANTS

Most of the members of the cruciferous crop plants are grown successfully in these regions. The cabbage is the most important and most widely grown, followed by the Chinese cabbage "wongbok," cauliflower, pechay, broccoli, radish, turnip, mustard, and Brussel sprouts.

DISEASES OF BROCCOLI AND BRUSSEL SPROUTS

Broccoli and Brussel sprouts are minor crops in the Mountain Province. Leaf spot, due to *Alternaria brassicæ* (Berk.) Sacc., is a common, but not a serious disease. The lower leaves are usually affected. The spots are usually light brown to dark brown with concentric rings.

DISEASES OF CABBAGE

Cabbage is the most extensively grown vegetable crop in Trinidad Valley and in other localities of the Mountain Province. As a single crop, the value of cabbage produced in the valley is estimated at several thousand pesos annually. The common diseases of cabbage are black leg [*Phoma lingam* (Tode) Desm.], black rot [*Plytomonas campestris* (Pammell) Bergey et al.], soft rot [*Erwinia carotovora* (Jones) Holland], and leaf spot [*Alternaria brassicæ* (Berk.) Sacc.].

Black leg (*Phoma lingam*).—In 1929, this disease was observed as common on most farms in Trinidad Valley, Lucban, Quisad, and Camp 7, causing 5 to 20 per cent infection. Since then, it has increased in severity and distribution, having appeared in almost every farm in Trinidad Valley. In September, 1932, it was found at Haight's Place, Mountain Trail, 52 kilometers

[3] The first plant-disease survey was started late in 1929 and has been continued from time to time up to the present.

from Baguio, where cabbage and other garden crops are also commercially grown. The disease is present all the year around, but most prevalent during the early and late parts of the season, when the weather is warm and moist. The fungus attacks plants at any stage of growth and causes blackening or rotting of the stem or spotting of the leaves (Plate 1 and Plate 2, fig. 1). As a result of the infection on the stem the disease in the early stages causes the lower leaves of the infected plants to turn bluish red, but as the rotting on the stem progresses, the plant gradually wilts and finally dies; if, however, the infection on the stem is slight, the plant may fail to form a head. From the infected stems and leaves, pustules (pycnidia) of the fungus are developed (Plate 1, figs. 1 and 2, and Plate 2, fig. 1).

The cabbage varieties commonly grown and susceptible to the disease are Early Market, Flat Dutch, American Red, Succession, and Shanghai. Among these the Shanghai variety is most resistant to the disease. This variety, although successfully grown during the normal cabbage season, is generally planted when the crop is subjected to more rains than usual. It is a late variety and a heavy yielder. The organism is known to be carried on or in the seed, and undoubtedly the disease was introduced with the cabbage seeds some years ago from other countries where it is serious on cabbage. It is now established in this locality, and unless its spread is checked, it eventually may ruin the cabbage industry in the Mountain Province.

Black rot (*Phytomonas campestris*).—This is a common disease of cabbage and other cruciferous plants in these regions, where in some cases 100 per cent infection was observed in the field. The characteristic symptom of the disease is blackening of the veins from the margins of the leaves; later the tissues turn yellow and dry up, especially when the weather is dry (Plate 2, fig. 2). As the disease progresses, blackening of the veins of the vascular bundles extends downward to the stem, and from there infection follows the vascular system up and down the plant, thus infecting the other leaves, as is usually shown by the blackened veins of (some) inner leaves in the formed head. In some cases as a result of infection, the leaves turn yellow, wilt, and then fall off from the bottom up to the stem so that a diseased plant may show merely a bare stalk with tufts of leaves on top. Such plants are stunted and may fail to produce heads.

Contaminated soil, seed, manure, and plants are the chief carriers of *Phytomonas campestris*, and once introduced into the

field, the bacteria will long remain active in the soil and ready to infect cabbage or other cruciferous crops grown thereon. As the disease is carried with the seed it was probably introduced some years ago with contaminated seeds from abroad.

Soft rot (*Erwinia carotovora*).—This is a serious and a common disease of cabbage in the field, in transit, or in storage. It is recognized by the transformation of the affected part into soft, wet, pulpy masses, often giving off an offensive odor. All the cabbage varieties grown in the valley are susceptible to soft rot. This disease is more serious on crops harvested early or late in the season or on crops subjected to abundant moisture and warm temperature. Under such conditions, a big cabbage head may be rotted within a few days (Plate 2, fig. 4) and a loss of 10 to 25 per cent or more of the crop may be observed. In storage or shipment this trouble also seriously affects cabbage and other vegetables.

Leaf spot (*Alternaria brassicæ*).—This disease is found common in most cabbage farms in these regions (Plate 2, fig. 3). It causes spotting of the leaves with the characteristic black, circular, and concentric rings. It usually is not serious, as it generally affects the old leaves, but in neglected plots all except the inner leaves of the head become heavily spotted. In this case all or part of the leaves dry up. The spots vary from a dot to over 1 centimeter in diameter.

DISEASES OF CHINESE CABBAGE, "WONGBOK"

The Chinese cabbage, or "wongbok," is an important vegetable crop and is generally grown in Trinidad Valley. This crop is affected by certain diseases which give farmers much trouble.

Gray leaf spot (*Alternaria herculea Ell. and Mart.*).—The leaf-spot disease caused by *Alternaria herculea* is a very common and serious disease of "wongbok" in the greenhouse and in the field, causing 100 per cent infection. It is present all the year around, but most serious during warm, moist days, when the leaves become badly spotted (Plate 3). When these conditions are followed by warm, bright days, badly infected leaves dry up and become papery, as if the plant had been scorched by flame. In this case 20 to 50 per cent of the leaves may be culled from single mature plant with a head. The progress of the disease is somewhat checked during dry weather, when the infected tissues dry up and fall off, causing a shot-hole effect. Dry hot weather is not favorable to the disease, and a good crop may be obtained without the excessive use of protective spray. A simi-

lar disease was reported by Weimer in California where it is only of minor importance. Undoubtedly, this disease is also not indigenous in this country.

Soft rot (*Erwinia carotovora*), although a minor trouble of "wongbok," is common during warm weather. Damping-off is a common disease of seedlings in the greenhouse. *Rhizoctonia* species and *Pythium* species are the two fungi usually found in association with damping-off in the seedbeds in Trinidad Valley and in the vicinity of Baguio. Mosaic was found on "wongbok" in Trinidad Valley but was not common.

DISEASES OF CAULIFLOWER

The cauliflower, not as widely grown as cabbage or Chinese cabbage, is also subject to the common diseases of cabbage; namely, common leaf spot (*Alternaria brassicæ*), soft rot (*Erwinia carotovora*), and black leg (*Phoma lingam*).

The leaf-spot (*Alternaria brassicæ*) though common on the leaves and on the curd, is usually not serious (Plate 4). On the curd the fungus causes the infected regions to blacken and rot, and this tendency is encouraged by the method practiced in the valley for bleaching the curd. On the leaves the disease causes dark brown spots of varying size. Under neglected conditions it may cause serious spotting and drying up of a great portion of the leaves. This disease was observed also in Haight's Place, Mountain Province. Soft rot (*Erwinia carotovora*) is serious late in the season, when the weather is warm and moist. It is generally found in Trinidad, the vicinity of Baguio, and Haight's Place, Mountain Province. Black leg (*P. lingam*), also found on cauliflower, is of minor importance. This host seems to be more resistant than cabbage, as the infected plants suffer less.

DISEASES OF PECHAY

Pechay is usually grown as a crop for early or late season when it is too wet for successful growing of any other crop. The common diseases observed are the leaf spots caused either by *Cercospora brassicicola* Henn., or by a species of *Alternaria* [*Alternaria brassicæ* (Berk.) Sacc.?], white rust, caused by *Albugo candido* (Pers.) Ktz., and mosaic (virus). The two leaf-spot diseases caused by *Cercospora* and *Alternaria* are the important diseases of pechay, causing considerable spotting and dying of tissues on the leaves. The spots due to *Cercospora* are usually pale, papery, and slightly sunken (Plate 5, fig. 1),

while those due to *Alternaria* are usually characterized by light brown to brown spots with concentric rings (Plate 5, fig. 2). White rust (*Albugo candido*) is common but not generally serious. It affects the leaves, pods, or flower stalks, causing malformations on the infected regions. The disease is recognized by the presence of pustules (sori) with white masses of spores which are usually produced on the lower surface of the leaves or on the pods and stem. It was found also affecting shepherd's purse, radish, mustard, turnip, water cress, and a species of *Amaranthus*. Mosaic is not as serious on pechay in these regions as in the Chinese gardens in Manila, where 30 to 50 per cent infection or more was found.

DISEASES OF MUSTARD

Mustard is a minor crop. The variety usually grown is the Shanghai mustard. A leaf spot caused by *Alternaria* spp. was found attacking this plant. This disease is serious, especially on farms where spraying is not practiced.

DISEASES OF RADISH

The American red and the Chinese and Japanese white radishes are the ones generally grown, but on a limited scale. A leaf spot due to *Alternaria* sp. (*A. brassicæ?*) is the commonest disease found, although it is not usually serious on radishes. A disease that causes spotting and rotting of the fleshy root was observed but is generally not serious.

DISEASES OF TURNIP

Turnip is cultivated on a small scale. A leaf-spot disease due either to *Alternaria herculea* Ell. and Mart. or *Alternaria brassicæ* (Berk) Sacc. is common in Trinidad Valley and Haight's Place. These are generally minor diseases of turnip, but they become serious in neglected gardens (Plate 6).

DISEASES OF CUCURBITACEOUS CROP PLANTS

DISEASES OF CHAYOTE

The cucurbitaceous crop plants represented by chayote, cucumber, squash, and upo are not generally grown on as large a scale as the cruciferous plants.

Chayote, a recently introduced vegetable crop, is becoming more widely grown in Trinidad Valley, and in other localities

near Baguio. This crop has not been found affected with many diseases. A leaf spot of minor importance, due to *Cercospora,* has been found, most frequently on the older leaves.

DISEASES OF CUCUMBER

The American cucumber is grown commonly in Trinidad Valley, where it is generally planted in April and May. The downy mildew [*Peronoplasmopara cubensi* (B. and C.) Clinton] is the most serious disease of cucumber, causing 100 per cent infection in the field. This disease is serious during cold and moist weather (November to January and May and June), but less common during the drier months (February to April). It produces irregularly shaped yellowish spots on the surface of the leaves.

On the under surface of the leaves, the spots are brownish to purplish and spores of the fungus are abundantly produced. The affected leaves sooner or later dry up. The older leaves nearest the center of the hill show the worst drying, stunting the plants which in turn produce small, poorly developed fruits. The mosaic (virus) disease of cucumber, although frequently found, is not serious. Powdery mildew (*Erysiphe* spp.) was also found but is of minor importance on cucumber.

DISEASES OF SQUASH AND UPO

Squash and upo are minor crops grown only in small plots. Mosaic (virus) was found on upo and on American Summer, and native, squash but is usually not serious. The American Summer squash appeared to be more susceptible to mosaic than the native variety. The affected plants show marked puckering, mottling, and clearing of the leaves, and as a result the plants are stunted and fail to yield fruits. This is true especially in the case of plants infected severely while they are still young. Powdery mildew (*Erysiphe* spp.) is also found on native squash but does not cause very great damage.

DISEASES OF THE LEGUMINOUS CROP PLANTS

The leguminous crop plants are considered next in importance in the extent of cultivation to the cruciferous groups. The garden beans and peas are the two most important legume crops generally cultivated on most farms in this locality. Soybean and peanuts are cultivated only on a very limited scale.

DISEASES OF BEANS

The garden bean is generally grown throughout the season in Trinidad Valley and in other localities near Baguio. The angular leaf spot (*Isariopsis griseola* Sacc.), bean rust [*Uromyces appendiculatus* (Pers.) Lev.], anthracnose [*Colletotrichum lindemuthianum* (Sacc. and Mag.), Bri. and Cav.], stem rot (*Sclerotium rolfsii* Sacc.), damping-off (*Pythium* and *Rhizoctonia*), powdery mildew (*Erysiphe* spp.), and bean mosaic (virus) are commonly found on the bean.

Angular leaf spot (*Isariopsis griscola*).—This is a very serious disease affecting the leaves, stems, and pods. The lesions on the leaves are small angular brown spots usually delimited by the veins or veinlets (Plate 7, fig. 1). Numerous and severe infections on the leaves cause them sooner or later to dry up and fall off prematurely, defoliating the plant before it reaches the fruiting stage. The disease is serious during the early and late seasons when the weather is moist and warm. If infection is early and the weather favorable, plants become stunted or die and no pods may be gathered. Kentucky Wonder, Chinese Wax, Chinese black bean "luvias," Bountiful, Canada Wonder, and Genuine Cornfield were all found to be susceptible to the disease. The black bean, or luvias,[4] however, appeared to be more resistant than the other varieties. The fruiting structures of the fungus, which are small and black, are found in great numbers on the dead areas of the undersurface of the leaves. This disease is not endemic as it has not been found in other localities in Mountain Province.

The bean rust (*Uromyces appendiculatus*) is as serious on garden beans as the angular leaf spot. It is common, although most prevalent and serious during the drier months of the year. The rust pustules containing orange powdery masses are developed on any part of the plant, but they are more numerous on the leaves. When infection on the leaves is heavy, the plant is stunted or defoliated (Plate 8, fig. 1). All the varieties of the garden bean (*Phaseolus vulgaris* L.) grown in the locality are susceptible. Among the pole-bean varieties, the Chinese black bean, or luvias, is most resistant. The Kentucky Wonder is found to be very susceptible. In general the pole-bean var-

[4] This variety is commonly grown and has been cultivated in this region for some time. Its apparent natural resistance has perhaps been built up through continuous selection. The other varieties are of recent introduction.

ieties are more susceptible than the bush-bean varieties. No record of bean rust has heretofore been reported, but the disease is believed to have been introduced into the Islands.

Bean anthracnose (*Colletotrichum lindemuthianum*).—This disease was first noted in 1929 in Pacdal, and Guisad, Baguio, but in later survey infected pods were brought at various times by native farmers for sale in the markets of Baguio, indicating that the disease is more widely distributed in these regions.

The greatest damage is to the pods, which become unmarketable. It produces sunken dark brown to blackish spots which may be round or oval with a reddish margin, and when the weather is moist, pinkish spore masses develop from these spots (Plate 8, fig. 2). On the underside of the leaves, especially along the veins, it causes reddish brown to black-brown lesions, while on the seed yellowish to brown or black spots are produced. Plants at any stage of growth are attacked, and if infection occurs when the plants are young and under favorable conditions of cool moist weather, the disease may ruin the crop. As the fungus is known to be carried in the seed it was undoubtedly introduced into these regions some years ago with infected seeds.

Stem rot (*Sclerotium rolfsii*).—This fungus, causing rotting of the stem and finally death of the plant, was noted in 1930, but it has been found since then on various other garden crops in the agricultural school garden. It is recognized by the rotting of the stem, on which thick masses of white mycelia and small brown sclerotia about the size of mustard seeds are found. It is, however, still a minor disease on beans.

Damping-off (*Pythium and Rhizoctonia sp.*).—These are the two common fungi causing damping-off of young bean plants in the field. The extent of injury is more serious when beans are sown during the relatively wet season.

Powdery mildew (*Erysiphe spp.*).—This fungus is occasionally found on beans, but its effect is not as serious on them as on peas. It affects the leaves, pods, and stems, producing white powdery masses on the lesions. The affected leaves die prematurely, and the productiveness of the plant is greatly reduced. The worst damage, however, is the serious stunting of the plants or the rendering unmarketable of the infected pods. This disease becomes commoner during the drier months of the year.

Bean mosaic (virus) was observed in the valley in 1929 on the dwarf Japanese wax bean and on the pole Chinese black

bean where infection was estimated at 25 to 50 per cent. Mosaic plants are less productive, and under severe infection plants are stunted and may fail to produce pods. The disease is not yet serious but is becoming widespread in these regions.

General mottling, crinkling, cupping, or puckering symptoms on the leaves are characteristically produced during the warmer months (Plate 7, fig. 2), but when the weather becomes cooler and moister, these become less evident. The Chinese black and the Chinese wax beans showed more resistance to mosaic than any of the bean varieties grown in this region. This disease is known to be hereditary and, as it is carried in the seed, it has undoubtedly been introduced into these regions with the importation of foreign seeds. The spread of the disease to other localities is largely effected by insects and through infected seeds.

DISEASES OF THE PEA

Both the American and the Chinese "chicharo" peas are generally grown. The common diseases found on them are the ascochyta leaf blight [*Mycosphaerella pinodes* (B. and Bl.) Stone], powdery mildew (*Erysiphe* spp.), and tip burn (physiological).

The ascochyta leaf blight (*Mycosphaerella pinodes*) is the most serious disease of peas affecting plants at any stage of growth. It causes spotting or blotching and finally blighting or death of the leaves. When the weather is warm and moist, young severely infected plants die and the majority of the leaves of bearing plants dry up (Plate 9). The pods may be malformed or spotted, depending upon their age at the time of infection. The disease is most serious when the season is warm and moist, and this is especially true with crops sown early or late in the season when the weather is moister than usual. The American and the Chinese ("chicharo") peas are both susceptible to the attack of the disease, but the Chinese peas show more resistance than the American variety.

Powdery mildew (*Erysiphe* spp.) is a common disease of peas, affecting the leaves, stems, and pods and producing spots or lesions. This fungus is recognized by the white, powdery appearance of the affected parts. It is serious during March and April, when the season is usually dry. When infection is early and severe, the plants may die or become unproductive.

Tip burn is a physiological trouble of peas, usually developed on the tender leaves after a few days of rain followed by hot,

bright days. This trouble is not serious, but plants are set back by it.

DISEASES OF THE PEANUT

The peanut is not usually grown in the valley, but plants grown in test plots of Trinidad Agricultural School were found badly affected by leaf spot (*Septogloeum arachidis* Rac.). Seriously affected plants are heavily defoliated and greatly stunted in growth. This was the first time peanuts were planted at the agricultural school. Since it was learned that the original seeds were obtained from the lowlands, there is no doubt that the disease was introduced.

DISEASES OF THE SOY BEAN

The soy bean is not a regular crop in this region but was grown primarily for its value as a green manure. In the trial plots of the Bureau of Plant Industry and of the Trinidad Agricultural School the soy-bean rust (*Uromyces sojæ* Sydow) was common and caused considerable injury.

DISEASES OF SOLANACEOUS CROP PLANTS

The solanaceous crops cultivated in this vicinity such as potato, tomato, eggplant, and pepper are not as extensively grown as cabbage, garden beans, or garden peas, not because there is no market for them but because their culture has become less profitable due to the presence of serious diseases.

DISEASES OF THE EGGPLANT

The eggplant grows successfully, but is raised only on a limited scale. The Japanese eggplant is generally cultivated in Trinidad Valley. The leaf spot [*Phomopsis vexans* (Sacc. and Syd.) Harter], bacterial wilt [*Phytomonas solanaceara* (Erwin Smith) Bergey et al.], stem rot and fruit rot (*Sclerotium rolfsii* Sacc.), and cracking of fruits (physiological trouble) are the diseases found on the eggplant.

The leaf spot (*Phomopsis vexans*) was observed at Trinidad Agricultural School, causing 100 per cent infection in one of the eggplant plots. The disease causes considerable spotting and dying of the tissues, thus producing "shot-holes" on the leaves. During warm, moist days, severe field infection is noted, and infected plants may be completely defoliated. The American, the Japanese, and the Philippine eggplants are equally susceptible.

Stem rot (*Sclerotium rolfsii*), killing young and old plants, is usually found, although generally not a serious disease. This fungus has also been found rotting the fruits that were touching the ground. This disease is recognized by the presence of white masses of mycelia or of numerous small brown sclerotia on the infected stem or fruit. The bacterial wilt (*Phytomonas solanaceara*) was observed in 1930 to be serious in Camp 7, and in 1931 and 1932 it was found in Trinidad Valley. This trouble is recognized by the gradual wilting and final death of plants with no external indication of the disease on the stem or roots. When such plants are pulled off and the roots cut, a shiny bacterial ooze may be observed from the cut ends of the vascular bundles. Cracking of the fruit (physiological trouble) is common on eggplants that are subjected to frequent rains late in the season. This is a physiological trouble apparently due to excessive moisture.

DISEASES OF PEPPER

Both the sweet and the hot pepper varieties are grown. Mosaic (virus), stem rot (*Sclerotium rolfsii*), bacterial wilt [*Phytomonas solanaceara* (Erwin Smith) Bergey et al.], and fruit rots (due to *Bacteria, Alternaria, Macrosporium,* and *Phoma*) are the diseases found on pepper.

Mosaic (virus) is found to be quite general, causing 100 per cent infection in some fields. Mosaic plants showed various types and degrees of mottling and clearing symptoms. This disease affects the photosynthetic activity of the leaves; the loss in yield, therefore, is dependent upon the earliness of infection and the severity of leaf symptoms. The American pepper, the Mexican hot pepper, and the Philippine native peppers were found susceptible.

Stem rot (*Sclerotium rolfsii*), bacterial wilt (*Phytomonas solanaceara*), and fruit rots due to *Bacteria, Macrosporium, Alternaria,* and *Phoma,* even if found, are minor diseases of pepper at present.

DISEASES OF THE POTATO

The potato was once said to be the staple crop generally grown in Trinidad Valley and other localities in the Mountain Province. Because of certain diseases, however, potato culture is now limited and cannot be carried on with profit. The diseases found on potato are the late blight [*Phytophthora infestans* (Mont.) De Bary], potato scab [*Actinomyces scabies* (Thax.) Gussow], rhizoctonia (*Corticium vagum* B and C.), early blight [*Alter-*

naria solani (E. and M.) Jones and Grout], and storage rots (fusarial and bacterial species).

The late blight (*Phytophthora infestans*) is the most destructive disease of the potato (also tomato) in the Mountain Province but has not yet been observed in the lowland provinces. It was noted in 1929, but it is believed that it has been in Trinidad Valley (4,263 feet) and in the vicinity of Baguio (4,989 feet) for some years.[5] In a limited survey the disease was also found in Antamok, 2,500 to 3,000 feet elevation; at Acop's Place, 4,300 to 4,500 feet; and at Haight's Place, Mount Paoay, Benguet, 7,350 to 8,000 feet, indicating that the disease is now widely distributed in the mountain regions, where the altitude is high.

The fungus attacks the tubers and all aërial parts of the plant. The lesions on the tubers are characterized by slightly shrunken, purplish brown spots. On the leaves the disease manifests itself as water-soaked brown lesions, usually starting from the margin or edge of the leaflets, and gradually or rapidly involve the whole leaf, depending upon the weather conditions. On the stem identical brownish lesions are produced. From the lesions on the leaves, stems, or tubers the fruiting bodies of the fungus, appearing as "white tuffs," are developed. When the weather is dry or sunny the progress of the disease is retarded and the infected leaves shrivel up, but when the weather is cool and moist, especially when there are a few days of rain, it spreads rapidly, so that the infected leaves, stems, and other parts are killed within a few days. Under these favorable weather conditions the whole potato field may be ruined in less than a week (Plate 10).

The late blight organism is known to be carried with infected tubers and undoubtedly was introduced into the Philippines from abroad. Its presence in other localities of the Mountain Province obviously is due to the use of contaminated tubers. The spores of the fungus are developed in great numbers and readily carried by wind, water, and other means, which may account for the rapid spread of the disease during favorable conditions in the field.

Potato scab (*Actinomyces scabies*) is quite prevalent in most farms in Trinidad Valley. From field counts made of small potato plots in 1932 at Kilometer 4, Trinidad Valley, about 50

[5] As this disease is usually serious after a few days of rain, the farmers of this region usually blame the rain as the cause of their potato crop failure.

per cent of the crop showed scab infection. The disease is characterized by spots or scabbed lesions, which disfigure the appearance of the tubers (Plate 11, fig. 4). All potatoes grown in Trinidad Valley are susceptible, but the red-skinned ones appeared to be less affected by the disease. The fungus is carried with the tubers and no doubt it also was introduced from abroad.

The rhizoctonia disease (*Corticium vagum*) has been found common on both native and imported potatoes offered for sale in the Baguio market. Infected tubers may be recognized by the presence of black sclerotia or "dirt which does not wash off" on the surface of the potato (Plate 11, fig. 3). When infected tubers are used for planting, the sclerotia of the fungus become active and attack and kill the young sprouts as they come up or produce sunken colored lesions on the stems. Plants with lesions on the stem are generally weak; and because of the injury on the stem the tubers, which are normally produced underground, may develop aërially along the stem.

This disease does not yet assume as important proportions as other maladies mentioned above, but in other countries, the rhizoctonia disease is considered very important on potato. The causal organism is carried as "sclerotia" on the tubers, and no doubt it has been introduced into the Philippines in this way. Once the organism is introduced, it remains active in the soil for many years, either living as a saprophyte on dead tissues or as a parasite on other living susceptible plants.

Mosaic (*virus*).—The mild and rugose type of mosaic is widespread and is found in many farms in Trinidad Valley and in various places near Baguio. In some farms it is not uncommon to find potato fields with 10 to 25 per cent infection from infected tubers. In 1932 it was found on volunteer plants at Haight's Place and on one of the gardens at Antamok Gold Field. Mosaic plants show the typical mottling, crinkling, or puckering symptoms of the leaves. This disease is one of the most serious diseases of potato in the United States, but it is not yet a serious problem on potatoes in this region. It is perpetuated in diseased tubers, and its occurrence in the Philippines must be traced to imported diseased tubers. Insects (aphids) are known to transmit it to healthy plants in the field.

Stem rot (*Sclerotium rolfsii.*)—This fungus causes rotting of the stem near the soil line, and the death of the plant results. This fungus is also found causing rotting of tubers in the field or in storage. It is recognized by the presence of white masses

of mycelia or of round brownish sclerotia about the size of a mustard seed on or near the infected regions. Other plants besides the potato have been observed affected by this disease.

Early blight (*Alternaria solani*) was found at Guisad, Baguio, in Trinidad Valley, and in Haight's Place, Mountain Province. It appears only occasionally, and when present causes little injury. It may be recognized by brown circular or oval spots on the leaves, which may show concentric rings.

Soft rot (*Erwinia carotovora*) and dry rot (*Fusarium* sp.), only of minor importance at present, are common storage troubles of the potato.

DISEASES OF THE TOMATO

The tomato grows well in the Mountain Province and has commercial possibilities. As in the case of potatoes, however, because of certain diseases its culture is now limited to the extent of being often omitted in the seasonal rotation of crops. The diseases commonly found are late blight [*Phytophthora infestans* (Mont.) De Bary], tomato wilt [*Phytomonas solanaceara* (Erwin Smith) Bergey et al.], mosaic (virus), top wilt and spot necrosis (cause unknown), fern leaf (virus), powdery mildew (*Erysiphe* sp.), root knot [*Heterodera radicicola* (Greef) Muller], stem rot (*Sclerotium rolfsii* Sac.), fruit rots (bacterial, fusarial, and sclerotial causes), and early blight [*Alternaria solani* (E. and M.) Jones and Grout].

The late blight (*Phytophthora infestans*) is also a serious disease of tomato as on potato, and the limiting factor in the successful cultivation of tomato in Trinidad Valley and in other localities of Baguio, Mountain Province. This disease assumes epiphytotic severity when the weather is cool and wet, ruining the whole field in a few days. All the vegetative aërial parts of the plant, including the fruit, are affected by the disease (Plate 12, and Plate 14, figs. 1 and 2). The symptoms of the disease on the aërial parts are almost identical with those found on the potato, while on the fruits the lesions are usually light brown, firm, and somewhat sunken. Under severe infection late in the season from 50 to 80 per cent of the fruit may be ruined by the disease (Plate 14, figs. 1 and 2).

The Giant Ponderosa, Dwarf Champion, New York Dwarf Champion, and the native varieties that are commonly grown are found very susceptible to the blight. The disease has been observed since 1929, although it is believed to have been here some

years ago. Whether the tomato and potato are affected by the same fungus cannot be definitely stated at present, but studies on these two late blight diseases are in progress.

Tomato wilt (*Phytomonas solanaceara*) was noted on a few tomato plants at Trinidad Agricultural School during the season of 1929–30. In 1932 it was observed to be more serious on the same plot, 50 per cent or more of the tomato plants having wilted and died due to wilt. This disease is serious in the tomato-growing regions in the lowland provinces; and, no doubt, may also become an important disease of the tomato in these regions. The infected plants gradually wilt, and finally die. No tomato variety is yet known to be resistant to this disease.

Mosaic (virus) on tomato is usually not a serious disease in the field in Trinidad Valley or in the tomato fields in the lowland provinces. A serious outbreak of mosaic, however, was observed in 1932–33 in the greenhouse at Trinidad Agricultural School, where all the commercial American and Philippine tomatoes showed 100 per cent infection. Plants infected showed typical mottling and clearing of the leaves (Plate 13, fig. 1).

Top wilt and spot necrosis (cause unknown).—A new malady not yet observed in the field was noted in November, 1932, at the time when mosaic was found in the greenhouse of Trinidad Agricultural School. The disease manifests itself either as small necrotic areas (spots) on the young leaves of the infected shoot, or such leaves become light green, stiff, gradually wilt and then finally dry up, leaving the older leaves of the shoot unaffected.

The disease is considered more serious than that of mosaic (virus) as it causes not only the young shoots but also the flowers to dry up and fall. All the varieties of tomato grown in Trinidad are susceptible. No organism causing the trouble has yet been isolated. Although in certain stages the disease shows some of the symptoms of the Australian spotted wilt disease of tomato, it cannot be considered as the same until further investigations have been made.

Fern leaf (virus), a minor disease of tomato (Plate 13, fig. 2), was noted in Antamok, Baguio, and in the greenhouse of Trinidad Agricultural School. This is also a minor disease of tomato in the lowland provinces.

Powdery mildew (*Erysiphe* spp.) is rarely found on tomato in the field. A serious outbreak of the disease, however, was observed in 1932 in the greenhouse of Trinidad Agricultural School. It is characterized by the powdery white appearance of the sur-

face of the leaves. The affected leaves usually wilt and dry up, and plants infected with powdery mildew are commonly stunted.

The root knot (*Heterodera radicicola*) was found on a few plants in Trinidad Agricultural School and also in Camp 7, Baguio, Mountain Province. This disease is characterized by the swelling of the roots, and the stunting and yellowing of the plants. Because of the more favorable conditions, the disease may eventually become more serious in the glasshouses than in the field.

Stem rot (*Sclerotium rolfsii*), or southern blight, is a minor disease of tomato in Trinidad Valley. It attacks the stem, causing it to rot, and finally the plant wilts and dies. Beans, potatoes, and larkspur are other crops found to be infected by this fungus.

Fruit rots.—Rotting of tomato fruits in the field or in storage is due either to *Phytophthora infestans, Sclerotium rolfsii, Bacteria, Fusarium, Macrosporium,* or *Rhizopus. Phytophthora infestans* is the most serious, causing under adverse conditions from 50 to 80 per cent or more infection of fruits in the field. Such fruits are no longer salable (Plate 14, figs. 1 and 2). The rot due to *Phytophthora infestans* is usually firm, but may become soft and watery as a result of invasion by soft rotting organisms. Sclerotium rot (*Sclerotium rolfsii*) is usually found on fruits lying on or close to the ground, but it is not a serious disease (Plate 14, fig. 3). The other fruit rots due to *Bacteria, Fusarium, Macrosporium,* and *Rhizopus,* of minor importance, are occasionally found in the field or in storage at room temperature.

The early blight (*Alternaria solani*) is a minor disease of tomato found in May, 1932, in Guisad, where older leaves close to the ground were most severely attacked.

DISEASES OF UMBELLIFEROUS CROP PLANTS

Among the umbelliferous crop plants, carrots and celery are the two important crops grown commercially in these regions. Their culture, however, is not as extensive and general as that of cabbage, wongbok, beans, and peas.

DISEASES OF THE CARROT

The carrot is a common crop in Trinidad Valley. The leaf blight (*Macrosporium carotæ* E. and E.) is the most serious disease found on the carrot. It causes spotting and blighting of the leaves so that the whole top may be completely blighted or blackened (Plate 15, figs. 1 and 2). It is much more serious

during the early and late seasons when the weather is warm and rains are frequent. During the dry season, from December to April, the disease is less severe so that a good crop may be obtained without the use of protective sprays.

DISEASES OF CELERY

Celery is generally grown, but the presence of certain maladies often discourages farmers from planting it on a large scale. The diseases found are the late blight [*Septoria apii* (Br. and Cav.) Rostrup], black-heart (physiological), soft rot (*Erwinia carotovora*), root knot [*Heterodera radicicola* (Greef) Muller], dwarf (virus), and yellows (virus).

The late blight (*Septoria apii*) is a serious and widespread disease of celery in Trinidad Valley and the environs of Baguio. It is present all the year around in the greenhouse or in the field, but it is more serious during the early and late season, when the weather is warm and moist, severely blighting or spotting the leaves and leaf stalks (Plates 16 and 17). Plants severely infected are stunted and dwarfed so that they become unmarketable. The small spot type of late blight appeared to be the most prevalent and serious in this region and is recognized by the appearance of small irregular spots which are studded with numerous black fruiting bodies (pycnidia) of the fungus (Plates 16 and 17). The blanching method often employed in these regions, where plants are inclosed in wooden boxes or the stems covered with grass, is conducive to late blight infection. The American Self Blanching and the White Plume are the common varieties grown, and both are very susceptible.

Black heart (physiological) is common and rather serious on late plantings. In May, 1932, 100 per cent infection was found on celery plants growing at the farm of Trinidad Agricultural School. The disease may cause only slight tipburn on one or more of the inner younger heart leaves, but in the worst cases the entire heart is killed and turns black (Plate 18). Because of invasion of secondary organisms, the "diseased heart" may be completely rotted.

Soft rot (*Erwinia carotovora*).—A complete rotting of the "heart" without the intervention of the black-heart disease was also noted to be serious on celery. This is caused by the invasion of a bacterium, *Erwinia carotovora*. This disease is most active late in the season when the weather is warm and rains are frequent. Where straw and grass are used for blanching,

soft rot is more prevalent. Slugs, which are more abundant under these conditions, aid in spreading the soft-rot organism.

The root-knot nematode (*Heterodera radicicola*) was found on celery plants in glasshouse seed beds in Camp 7, Baguio. It was not as serious in the field as it is in seed beds under glasshouses. This disease is characterized by the swelling of the roots, yellowing of the leaves, and stunting of the plants.

Dwarf (virus) and mosaic (virus) diseases were found in 1930 in the Japanese Bazaar garden in Trinidad Valley. The dwarf disease causes dwarfing of the plant. The stalks are very much shortened and crowded and the plants are greatly stunted (Plate 19). Besides exhibiting some of the above symptoms, the mosaic disease is characterized by the mottling, clearing, or puckering of the leaves. These two virus diseases are of minor importance on celery.

DISEASES OF MISCELLANEOUS CROP PLANTS

DISEASES OF THE BEET

The beet is generally grown for leaves and for the root. The common leaf spot (*Cercospora beticola* Sacc.) and root canker (*Rhizoctonia* spp. or *Fusarium* spp?) are the two important diseases of the beet plant.

The leaf spot (*Cercospora beticola*) is very widespread in the field and in the greenhouse, causing 100 per cent infection. During warm and moist weather, especially in the early and late parts of the season, considerable spotting and rotting of the leaves are noted. When the weather is dry, the progress of the disease is checked; the infected tissues dry up and drop off, badly perforating the leaves. The disease is present the year around, but less serious during the dry season, so that a good crop may be harvested without the use of sprays.

Root canker (*Rhizoctonia* spp. and *Fusarium* spp.?) on beets was observed to be serious in 1930 at Trinidad Agricultural School, badly cracking or corroding 25 to 75 per cent of the crop (Plate 20), making it less salable. The disease is most prevalent on beets planted in December or January and harvested during the warmer months. Infected plants are usually dwarfed, and the petioles of the young leaves turn black and finally die. The first sign of the disease on the fleshy tap root is slight browning or blackening of the affected region, while in the more-advanced stages cracking or corroding of a greater portion of the tissues may be observed. Isolations made from the fresh or advanced lesions showed that species of both *Rhizoctonia*

and *Fusarium* are associated with the malady. Thus far, however, no test has been made to show which of these organisms is the real cause of the trouble.

DISEASES OF CORN

Philippine native corn and American sweet corn are usually cultivated as a garden crop during March, April, and May. Corn rust (*Puccinia sorghi* Schw.) is a disease very commonly observed in Trinidad Valley but not in the corn-growing regions in the lowland provinces. It was also observed in 1930 on a farm in Lilio, Laguna Province, Luzon, at the foot of Mount Banahao, where the climate is relatively cool and moist. The disease is recognized by rust pustules on the leaves and leaf sheaths containing orange yellow powdery-spore masses. When plants are infected early and numerous rust pustules are developed, part or all of the leaves dry up and as a result the plants are dwarfed. Both native corn and American sweet corn grown in Trinidad Valley were found to be very susceptible to the disease.

DISEASES OF LETTUCE

Both leaf lettuce and head lettuce are grown in these regions. The important diseases noted on them are leaf spot (*Cercospora lactucæ* Stevenson), soft rot (*Erwinia carotovora*), and tipburn (physiological trouble).

Leaf spot (*Cercospora lactucæ*) is a common disease late in the season, especially severely affecting the flowering lettuce plants. The spots are brown, round to irregular in shape, and characterized by a dead gray center which is surrounded by a light brown infected area. It is most serious during the warm moist weather of May, June, and July, when 25 to 100 per cent of the leaves may be badly spotted. This disease is also found to be serious on lettuce in the gardens around Manila.

Tipburn, a physiological trouble in which excessive water and rapid evaporation may be associated, was found to be serious on lettuce grown on the reclaimed plots in the swamp in Trinidad Valley. A certain variety with yellowish, pale, thin leaves showed 100 per cent tipburn, while the "Los Angeles," a dark green thick-leaf variety, showed less than 10 per cent infection. The trouble is commonest during April and the early part of May when the weather is usually warm and dry. The symptoms are limited to "burning" of the edges and tips of the leaves (Plate 21), but because of secondary invasion of other organisms the infected parts rotted rapidly.

The soft rot (*Erwinia carotovora*) is also found on this plant but is not a serious trouble of lettuce in these regions.

DISEASES OF OATS, WHEAT, AND BARLEY

Temperate-zone cereal crops such as oats, wheat, and barley have been found to grow successfully in the Mountain Province. In Trinidad Valley, wheat and barley were found growing well and free from serious diseases. The loose smut [*Ustilago avenæ* (Pers.) Jens.] was found at Haight's Place, Mountain Province, where five panicles from a small plot planted with oats showed smut infection. The disease manifests itself by the complete transformation of grains into black powdery masses which are the spores of the fungus (Plate 22). The causal organism is known to be carried with the seed, and no doubt its first occurrence in Haight's Place is due to the presence of contaminated seeds in the lot. From information it was found that the seeds were obtained from England the year before, and no doubt the disease has been introduced.

DISEASES OF THE ONION

The onion is grown more extensively in Trinidad Valley for its top than for the dried onion bulb. A leaf blight of the green onion caused by *Macrosporium porri* Ellis has been found to be most serious, causing 100 per cent infection in the field. Under favorable environmental conditions of warmth and moisture the disease causes complete blighting of the leaves (Plates 23 and 24). The white Bermuda onion and the Chinese green onion are both susceptible. Garlic, "kutchay," and other members of this family, which are grown for their tops but on a very small scale, have not been found to be affected by the disease.

DISEASES OF RHUBARB

Rhubarb is still grown on small plots, but it is becoming more popular among the Chinese and Japanese farmers. The rhubarb rust [*Puccinia phragmitis* (Schum.) Korn.] has been found in Trinidad Valley and at Haight's Place, Mountain Province. The disease is usually severe on the lower leaves. The spots are small, but when they coalesce bigger spots are formed. The disease is seldom serious.

DISEASES OF THE STRAWBERRY

The strawberry constitutes a favorite crop among the native farmers in Trinidad Valley and other localities near Baguio. It is not, however, as extensively cultivated as the cabbage. The

diseases found on the strawberry are leaf spot [*Mycosphærella fragariæ* (Schw.) Lind.], fruit rot (*Rhizopus nigricans* Erh.), dwarf (virus), and yellows (virus).

Leaf spot (*Mycosphærella fragariæ*).—This disease appears to be common and is generally found on nearly all strawberry plots in Baguio, in Trinidad Valley, and, at Haight's Place, Mountain Province. In certain cases the spots are so numerous as to cause considerable "shot-holes" and thus to stunt the plants (Plate 25). All the common commercial varieties and the native ones grown in these regions are susceptible to the disease.

Fruit rot.—The black mold (*Rhizopus nigricans*) is a common fungus, causing considerable rotting of strawberry fruits in the field but especially in storage and transit. This disease is recognized as a soft watery rot, but in advanced stages the infected fruit may be completely rotted and mushy, and covered by the mycelia and black fruiting bodies of the fungus (Plate 26). Shipment of strawberries from Baguio to Manila markets and storage without refrigeration often result in a loss of 30 to 50 per cent or more due to this fruit rot. The fungus is favored by warm temperature (room temperature), and because of improper handling and lack of refrigeration the disease, once started, progresses so rapidly that a basketful of strawberries may be completely ruined in one or two days (Plate 26).

Strawberry dwarf, strawberry yellows (virus diseases), and stem canker (*Rhizoctonia*) are other less important diseases of the strawberry.

GENERAL DISCUSSION

By no means all diseases on the various crops in Trinidad Valley and the vicinity of Baguio, Mountain Province, are here reported. Others may have been present when the surveys were made. A few of these diseases are tropical, but many of them are representatively semitemperate or temperate in habitat, probably of foreign origin, introduced (with seeds, vegetative parts, etc.) recently or some years ago and here reported for the first time in this part of the Philippines. Some of them are now limiting the culture of certain crops, and even those that at present are of minor importance may in a few years become formidable obstacles to successful farming in these localities. Some of these diseases, such as the late blight of potato and tomato, late blight of celery, the leaf spot of "wongbok" and pechay, the angular leaf spot of beans, the bean rust, the asco-

chyta leaf blight of peas, the black leg of cabbage, and the soft rot of cabbage and other vegetables, are now serious and important, whereas a few years ago they were perhaps not of much consequence. Because of the ideal environment for the multiplication and the rapid spread of the organisms, the diseases found on the various crops are bound to increase and become a serious factor in these regions.

So far no detailed studies have been conducted for the purpose of controlling these diseases. Practical Japanese and Chinese farmers of Trinidad Valley and student farmers of Trinidad Agricultural School are making every effort to control some of them, but due to ignorance of the fundamental behavior of the diseases their experiences in most cases are costly and their results discouraging. Since conditions in the Philippines are different from those in other countries where the diseases have been studied, it will be necessary, before any intelligent, efficient, and specific control measures can be suggested for any particular disease, that information on the fundamental biological behavior of the organism and the epidemiology of the disease as it occurs in the Philippines be known. Knowledge of the biology of the host and information based on experiments in the use of protective sprays or dusts, and selection or breeding for resistance, should likewise be studied. Knowledge along these lines may eventually lead to change of farming practices in these regions.

Since the diseases listed above are caused by specific organisms and require specific control measures, and since effective control measures are usually based on experiments made under local conditions, what is effective in checking the disease elsewhere might prove useless under Philippine conditions. As time has not yet permitted the writer to study each of these diseases in detail, the following remedial and general control measures, if followed, may be of value, especially to the farmers in these localities.

Sanitation.—Most garden-crop diseases are either first introduced into the field with seeds, infected seedlings, or plant propagative parts, or brought in with the soil or with the wind. If the field conditions are favorable, the pathogenic organisms multiply rapidly and spread to various parts of the field by various agencies, such as insects, wind, water, contaminated soil clinging to the farm implements, hoofs of animals, feet of men, etc. Therefore, it is necessary to destroy by burning all infected plants and their parts found in the field or seedbed. Since

these serve as a source of infection to neighboring plants, the field should be kept free from all rubbish.

Healthy seeds.—Only seeds from healthy plants or seeds that are known to be free from diseases should be used. The seeds should be raised locally if practicable, or else purchased from reliable sources. Newly opened land or farms in isolated places should be protected from contamination with diseases brought in with seeds, seedlings, and other propagating stocks, or with contaminated soil.

Disinfection of seeds.—As diseases are carried on or in the seed, sterilizing the seeds with disinfectant is necessary. Mercuric chloride (1:1000) or formalin (40 per cent formaldehyde), diluted in water in the proportion of 1 pint to 30 gallons of water, is generally used as a disinfecting agent. The length of time for treatment depends upon the kind of seeds and the chemical used. Generally, small-seeded garden crops are sterilized after treatment of three to fifteen minutes, and for potato tubers one to two hours may be sufficient. For diseases carried internally by the seed or by the tubers, hot-water treatment, aging the seed for a few years, or the "index method" for tubers may be used.

Care of seed bed and seedlings.—The seed bed should be kept free from diseases. Young plants are subject to many diseases, and since they are important sources of infection, the seedlings transplanted to the field must be healthy. Neither care nor expense should be spared to protect them in the seed bed, whether one uses dusts or suitable sprays. The bed could be made free from diseases by sterilizing the soil with direct heat, steam, or chemicals, and if this is not possible, fresh soil should be used.

Spraying and dusting.—The use of sprays and dusts is one of the direct methods of control, but it is only temporary and its effectiveness largely depends upon thoroughness and frequency of application. It has for its purpose the protection of the plants with substances harmless to them and at the same time poisonous to the parasites. Standard Bordeaux mixture (4–4–50), Burgundy mixture, lime sulphur, flowers of sulphur, and copper-lime dust are some of the common sprays and dusts generally used to protect plants from diseases. The strength of the spray concentration or amount of dust applied or the frequency of their application depends upon the host plant and the disease under consideration.

Crop rotation.—Crop rotation is not only essential to the control of disease, but also beneficial to the maintenance of soil

fertility. The object of crop rotation as a means of control is to starve the parasites by changing the cultivation of a susceptible to that of a resistant host plant for two or more years. Plants of related species having diseases in common should not be planted in the same piece of ground continuously, or interplanted at the same season.

Resistant varieties.—The use of resistant varieties is the most profitable and most important phase in plant-disease control. The task of securing resistant or immune varieties is a long and arduous, but not an impossible one. There are crops now in cultivation that are resistant to certain serious diseases, which were developed by patient, continuous selection and breeding.

The selection and development of resistant varieties, however, has its limitations and cannot solve all difficulties. There are diseases against which resistance cannot be developed, and in such cases other means of control, such as spraying, dusting, and other methods mentioned above, should be used. Furthermore, one should bear in mind that in most cases resistance is specific to certain diseases only; it is local—that is, a plant may be immune in one locality and highly susceptible in another; and resistance in a certain crop may be temporary, and may disappear in the course of time. The variation or loss of resistance in such plants is not only due to the fact that plants vary in resistance to their parasites, but the parasites themselves vary in their ability to attack their host plants. Within these limitations the continued selection for resistance to disease and the use of resistant varieties when available and possible should therefore be encouraged, and once resistant crops have been obtained, they should be kept in pure line.

ILLUSTRATIONS

PLATE 1

Stems of cabbage plants infected by black leg (*Phoma lingam*). The extent of the injury, which caused the death of the plant, is shown. The pycnidia of the fungus may be seen at X. The plants died before the head was well developed.

PLATE 2

FIG. 1. A cabbage leaf taken from the lower old leaves of a plant infected with black leg.

2. Black rot on a cabbage leaf. The blackening of the veins advancing inward from the edge of the leaf, which is characteristic of the disease, is shown.
3. Leaf spot on a cabbage leaf. The lesions are circular to irregular spots, generally light brown to dark brown, with concentric rings.
4. A cabbage head infected with soft rot in the field and now showing an advanced stage of the disease. The central core is completely rotten and massy, emitting a foul odor.

PLATE 3

Chinese cabbage "wongbok" leaf, showing spots caused by *Alternaria herculea*. This leaf was taken from one of the other leaves of the formed head and is severely spotted.

PLATE 4

FIG. 1. Leaf spot of cauliflower, due to *Alternaria brassicæ*.

2. Head rot of cauliflower, due to *Altenaria brassicæ*.

PLATE 5

FIG. 1. Leaf spot of pechay, due to *Cercospora brassicicola*.

2. Leaf spot of pechay, due to *Alternaria* species, perhaps *Alternaria brassicæ*.

PLATE 6

Typical leaf spot of turnip, due to *Alternaria herculea*.

PLATE 7

FIG. 1. Angular leaf spot of garden bean, due to *Isariopsis griseola;* typical symptoms.

2. Mosaic (virus) of garden bean; typical symptoms.

PLATE 8

FIG. 1. Pustules of bean rust on a bean leaf.

2. Typical anthracnose spots on bean pods.

PLATE 9

Pea plants badly infected by ascochyta leaf blight. Most of the leaves on the lower portion of the stem are killed. The leaves and stem show typical lesions. Young plants severely affected are generally killed by this disease.

PLATE 10

A potato field infected with late blight disease. This field later became ruined.

PLATE 11

FIG. 1. Section of a healthy potato tuber.
2. Section of a potato tuber infected with potato blight, *Phytophthora infestans*. The characteristic browning and blackening of the tissues are shown.
3. Potato tubers infected with *Rhizoctonia*. The sclerotia of the fungus are seen adhering to the tuber.
4. A potato tuber showing typical scab lesions.
5. A healthy potato tuber.

PLATE 12

A portion of a tomato plot showing a tomato plant badly infected with late blight, *Phytophthora infestans*. Nearly all the aërial parts of the plant, including the fruits, showed infection. This plant died within a few days and failed to bear fruits.

PLATE 13

FIG. 1. Tomato leaves showing mosaic (virus) symptoms.
2. Tomato leaves showing fern leaf (virus) symptoms.

PLATE 14

FIG. 1. Fruit rot of tomato due to a late-blight organism. This fruit was later secondarily infected by a species of *Fusarium* (the white mass of mycelia), which caused complete rotting.
2. Section of a tomato fruit infected by late blight. The extensive browning of the tissues on the affected region, which is firm, is characteristic of the disease.
3. A tomato fruit infected by *Sclerotium rolfsii*. Young sclerotial bodies are formed on the infected region. Fruits close to the ground or lying on it are generally attacked by this fungus.

PLATE 15

FIG. 1. A carrot plant severely attacked by leaf blight (*Macrosporium carotæ*. The tops (leaves and petioles) are all killed or blighted. About × 0.5.
2. A dried carrot leaf; the condition is due to leaf blight. About × 2.

PLATE 16

Typical spots on the leaves and stem of celery badly infected by late blight, *Septoria apii*. The small spot type of late blight is more prevalent in this region.

PLATE 17

A "bleached" celery plant badly attacked by late blight, showing infection on the stems and the leaves. The outer and older leaves, because of the severe infection, are dried up. Only the few young leaves of the "heart" did not show infection.

PLATE 18

A celery plant infected with "black heart" disease. The entire heart of the plant turned black and died. Complete rotting of the "heart" may follow due to invasion of secondary organisms.

PLATE 19

A celery plant showing the typical symptoms of dwarf disease (virus). The crowding and shortening of the petioles of the infected plant are characteristic.

PLATE 20

Typical symptoms of dry rot on the beet. The corroded or cracked regions are characteristics results of the disease. A species of *Rhizoctonia* and a species of *Fusarium* are usually isolated from the lesions.

PLATE 21

FIG. 1. A lettuce leaf with dried or killed tip, due to tipburn.
2. A lettuce plant infected with tipburn. Some affected leaves can be readily recognized.
3. A lettuce leaf showing the typical spots due to *Cercospora lactucæ*.

PLATE 22

FIG. 1. A healthy oat panicle.
2. Oat panicle infected with loose smut. The grains are transformed into powdery black masses. These are the spores of the fungus.

PLATE 23

FIG. 1. Onion plants with dried infected leaves due to the leaf-blight disease (*Macrosporium porri*). About × 0.5.
2. An onion leaf showing the typical oval or irregular-shaped lesions. The black tufts in the lesions at X are the fruiting structures of the fungus.

PLATE 24

FIG. 1. A strawberry plant infected with the leaf-spot disease.
2. Typical lesions of the disease on the strawberry leaf.

PLATE 25

FIG. 1. A basket of healthy strawberry fruits, as marketed in Baguio.
2. A basket of strawberries completely ruined by common black mold (*Rhizopus nigricans*) after seventy-two hours at room temperature in the laboratory in Trinidad Agricultural School. The mycelia and the black fruiting bodies of the fungus may be seen covering the rotten fruit.

1 2

PLATE 1.

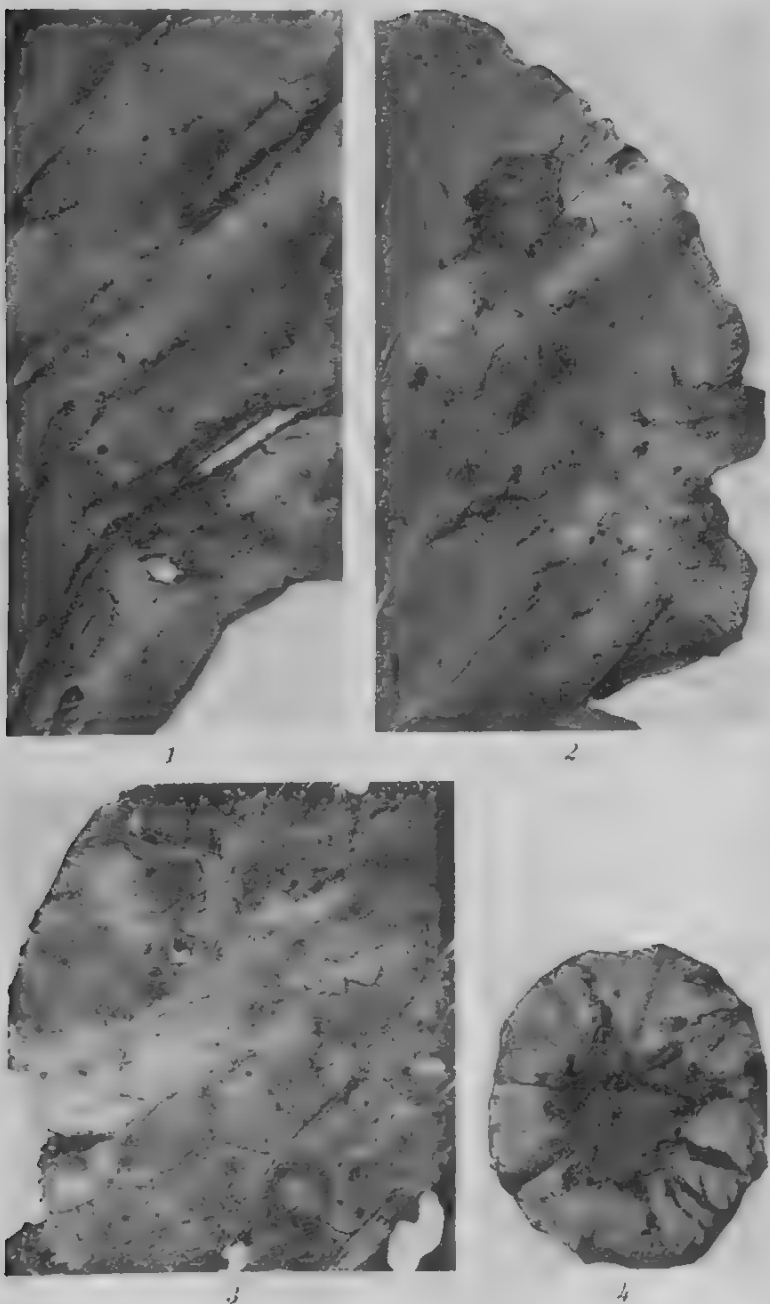
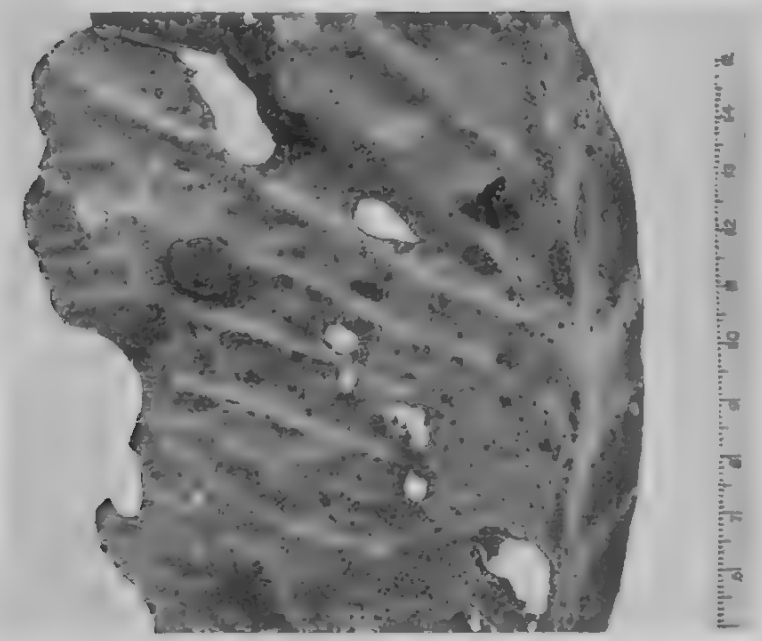


PLATE 2.

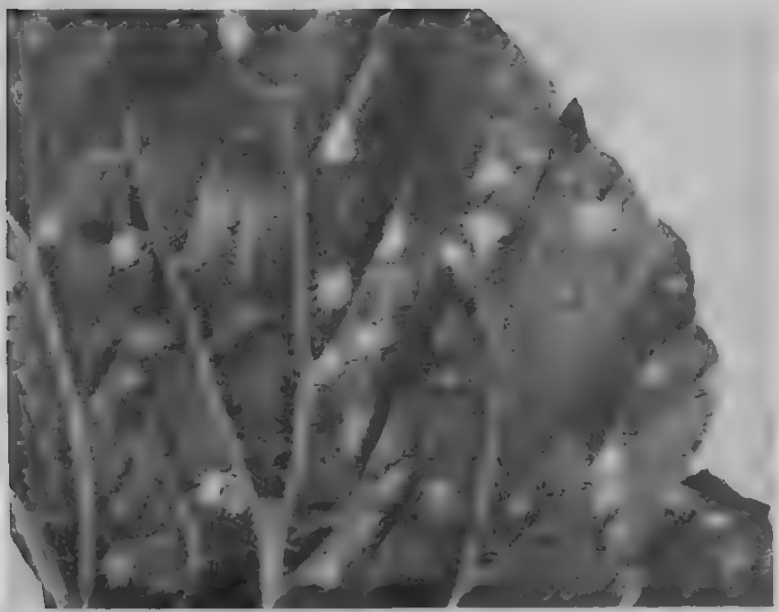
PLATE 3.



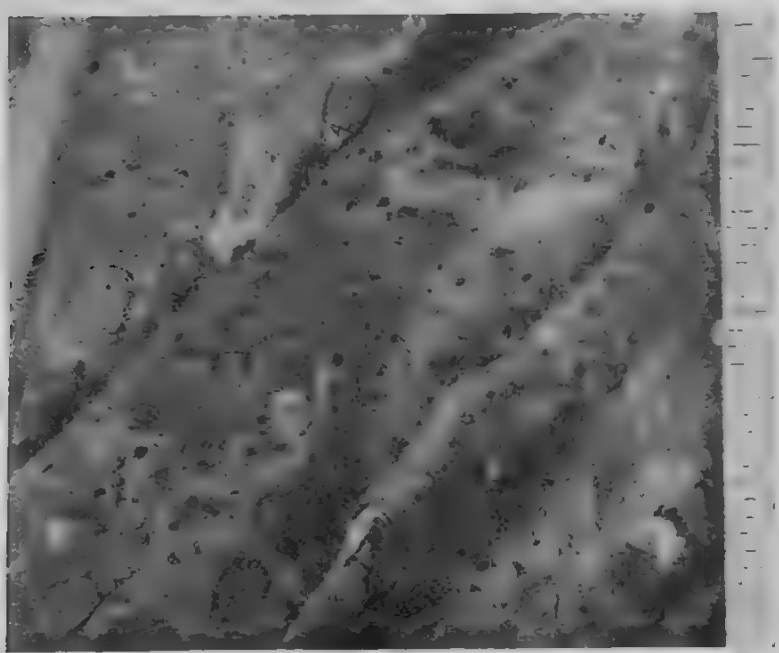
1

2 3

PLATE 4.



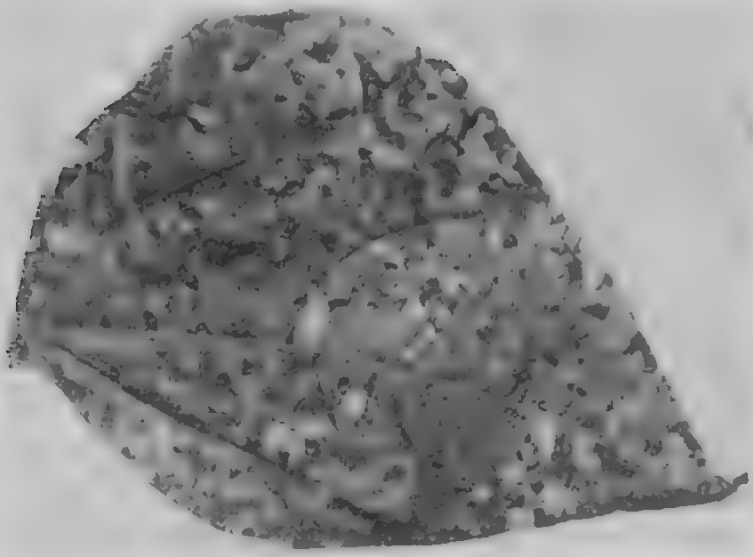
1



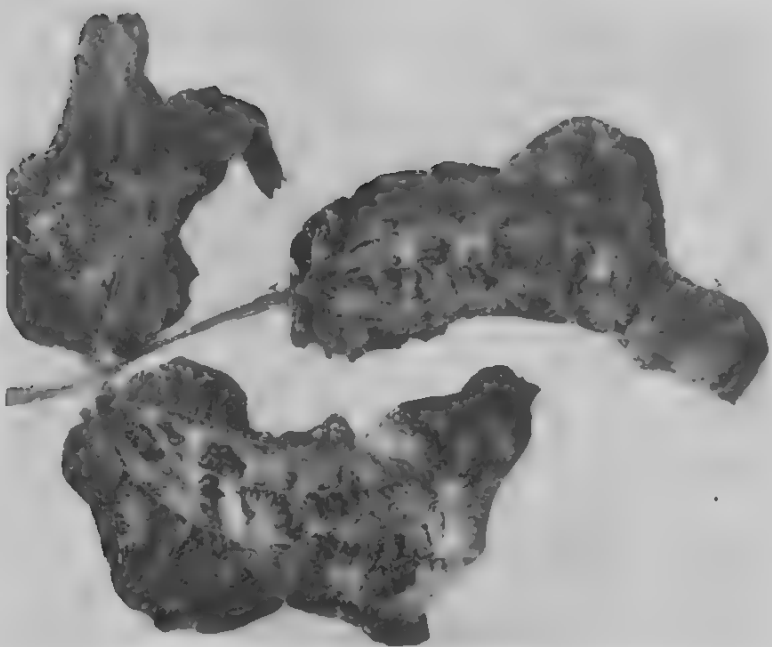
2

PLATE 5.

PLATE 6.



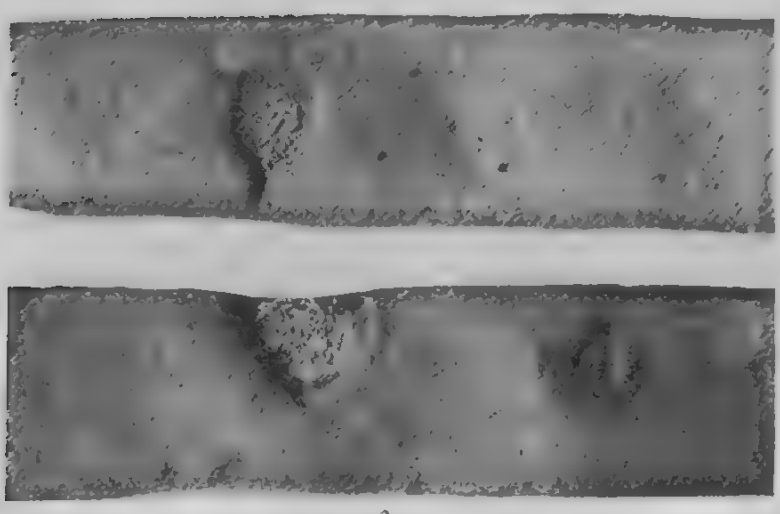
1



2

PLATE 7.

1



2

PLATE 8.

PLATE 9.

PLATE 10.

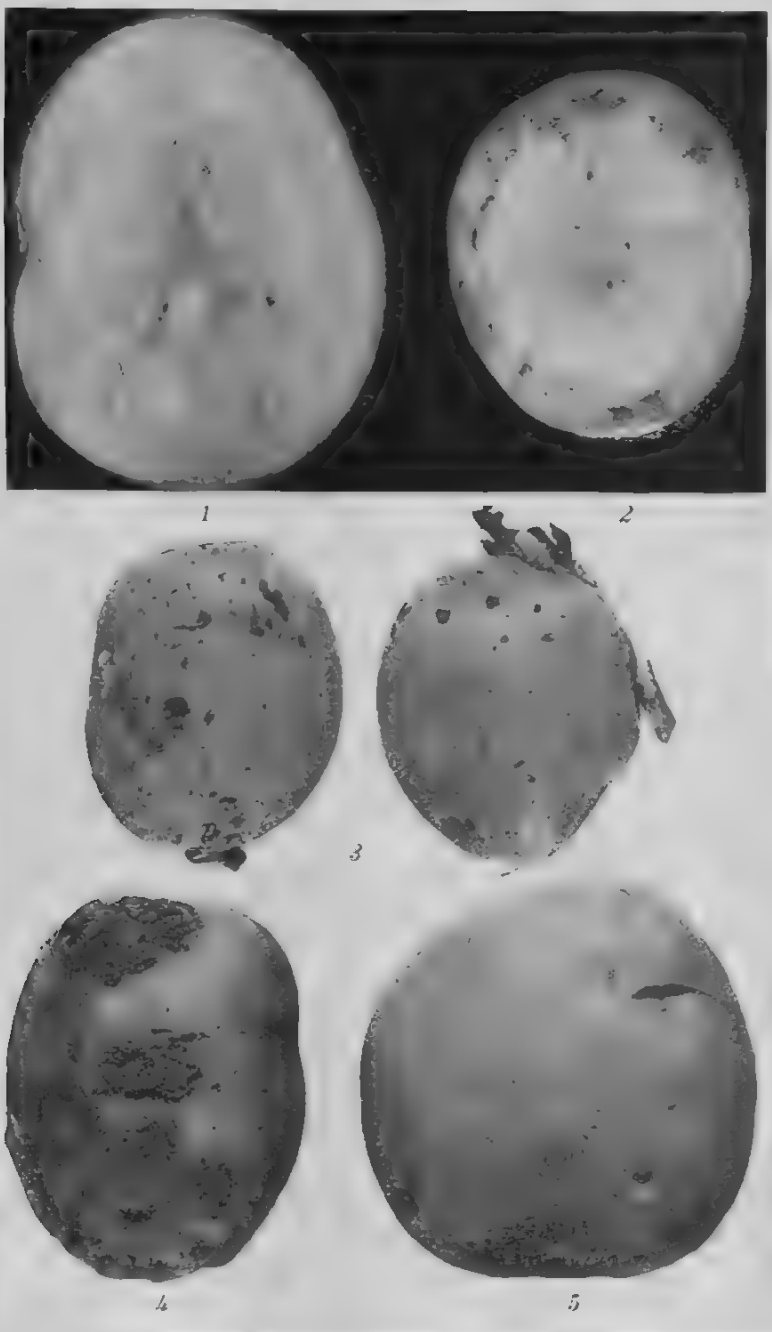
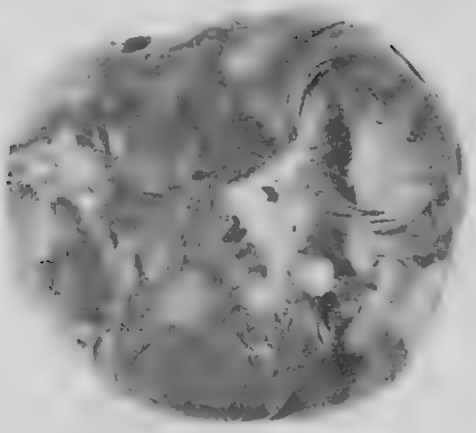


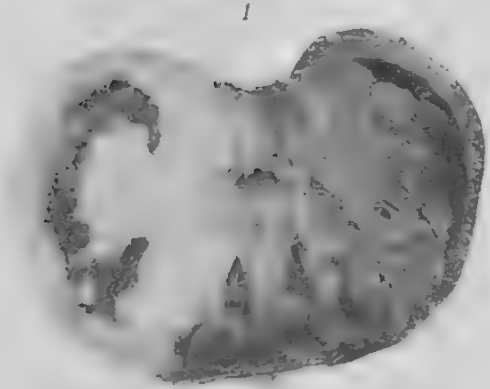
PLATE 11.

PLATE 12.

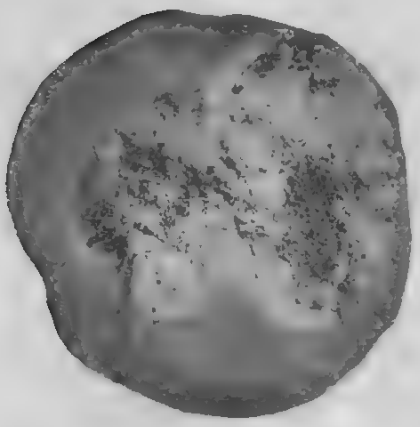
PLATE 13.



1



2



3

PLATE 14.

1 2

PLATE 15.

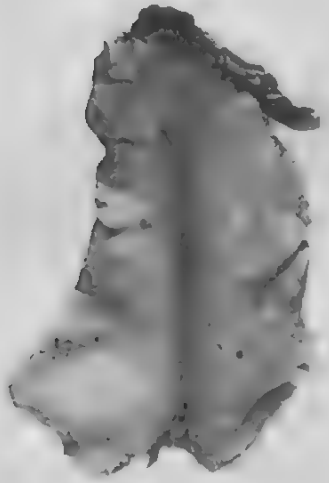
PLATE 16.

PLATE 17.

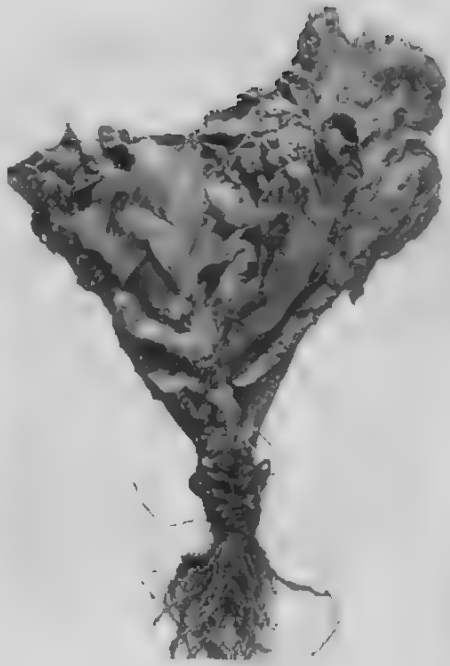
PLATE 18.

PLATE 19.

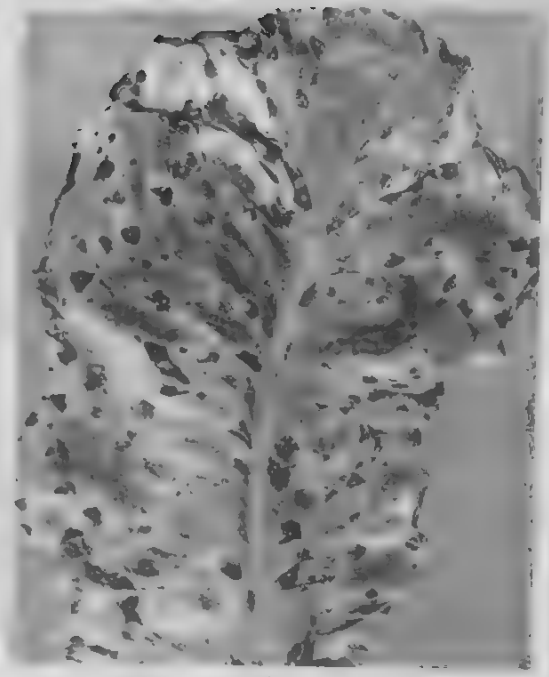
PLATE 20.



1



2



3

PLATE 21.

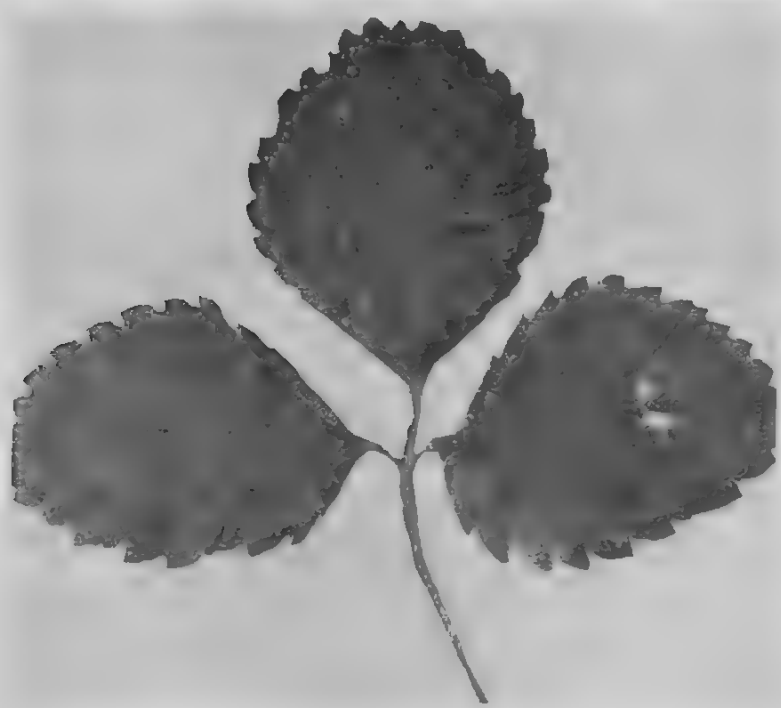
1 2

PLATE 22.

1 2

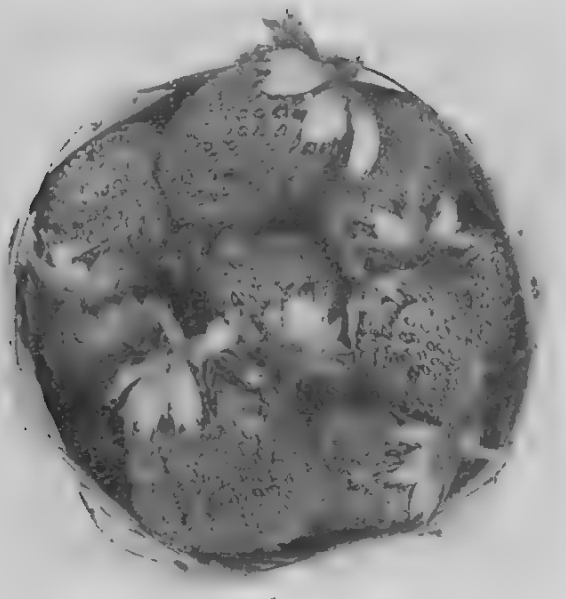
PLATE 23.

1

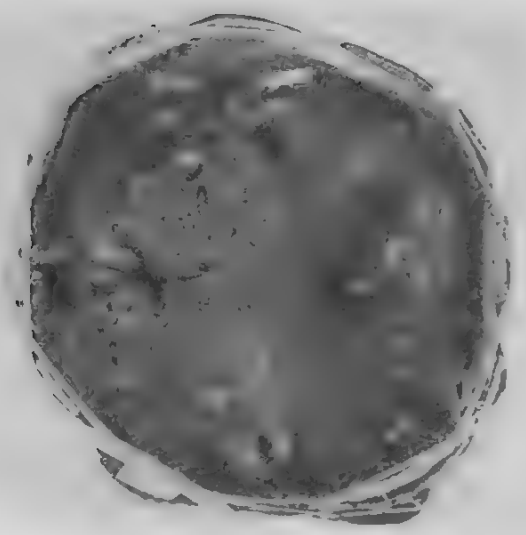


2

PLATE 24.



1



2

PLATE 25.

STUDIES IN SURRA, I

THE BLOOD CHEMISTRY IN EQUINE TRYPANOSOMIASIS (TRYPANOSOMA EVANSI)[1]

By RAYMOND RANDALL

Major, Veterinary Corps, United States Army
Member, United States Army Medical Department Research Board
Bureau of Science, Manila

The manner in which pathogenic trypanosomes produce death in their hosts has been a subject of considerable controversy. One method of approach in an attempt to solve the problem, which has been utilized by many workers, is by the study of the alterations of the blood chemistry in experimental trypanosomiasis.

Opposing views on the lethal factors in death from trypanosomiasis have been set forth as follows: First, that the injury and ultimate death of the host are due to a toxic substance liberated by the disintegration of the parasites, as proposed by Regendanz and Tropp (1927) and others; second, that the cause of injury and ultimate death of the host is the exhaustion of the blood sugar and glycogen reserve, as held by Schern (1925–28) and von Fenyvessy (1926); third, in rats at least, Andrews, Johnson, and Dormal (1930) believe that death of the host is due to asphyxia brought about by pulmonary œdema due to partial obstruction of the circulation by the agglutination of the trypanosomes in the heart and lungs.

Regendanz and Tropp, who support the toxin theory, were unable to confirm the findings of Schern and von Fenyvessy in regard to the exhaustion of blood sugar and glycogen reserve. Their results indicate that the glycogen depletion is by no means complete and at the height of the infection sufficient glycogen remains in the liver to maintain a normal sugar concentration in the blood. They maintain that the decrease in the blood sugar is due to a depressive effect on sugar inversion by the trypanosome toxin and not the depletion of glycogen.

[1] This paper was read in part before the Fifteenth Annual Meeting of the Philippine Veterinary Medical Association February 11, 1933, and the completed manuscript submitted for publication October, 1933.

Kligler, Geiger, and Comaroff (1929), investigating von Fenyvessy's toxin theory, injected massive doses of autolyzed trypanosomes and plasma respectively from guinea pigs heavily infected with various pathogenic trypanosomes into rabbits and rats, the animals receiving from seven to ten injections in the course of two weeks. In the rabbits the injection of autolyzed trypanosomes and, to some extent, the injection of plasma alone produced a leucopenia. As no other toxic manifestation was observed in either the rats or rabbits, they conclude that the repeated injection of heavy doses of autolyzed trypanosomes did not exert an appreciable toxic effect on the animals. The effect produced, as shown by the leucopenia, is not sufficient to account for the severe damage and ultimate death caused by the infection on the basis of a toxæmia. These workers also made quantitative determinations of the lactic acid in the blood and of the oxygen consumption of rats infected with *Trypanosoma evansi*. They did not find a higher oxygen consumption in infected rats and assume that incompletely oxidized metabolic products of glucose from the activity of the trypanosomes produce a state of acidosis in the animal, leading to exhaustion of the alkali reserve and probably also to a depression of the oxidative processes by the specific effect of lactic acid on the hæmoglobin. By supplementing the alkali reserve twice daily with 0.5 cc of 10 per cent sodium bicarbonate solution, intraperitoneally, they were able to prolong the life of a treated set of rats 50 per cent over that of an untreated set.

Scheff (1928, 1932) agrees that trypanosomes utilize large amounts of glucose and states that the glycogen reserve of the liver becomes exhausted. In guinea pigs, in which the course of infection is prolonged and undulating as compared to the rat, the resulting hypoglycæmia is apparently compensated as shown by a hyperlipemia. He describes in rats infected with *Trypanosoma equiperdum*, a form of internal asphyxia further embarrassing the liver, which is followed by an acidosis contributing to the metabolic disturbances leading to the death of the animal.

Andrews and his associates, using rats infected with *Trypanosoma equiperdum*, state that the animals die of asphyxia and on pathologic evidence, conclude that this is due to a retardation in the circulation of the blood due to agglutination of trypanosomes in the heart and lungs. The consequent anoxæmia leads to a nonvolatile uncompensated acidosis and to central necrosis of the liver, interfering with both its glycogenic and glycogenolytic functions and ultimately producing a hypoglycæmia.

STUDIES IN SURRA, I

THE BLOOD CHEMISTRY IN EQUINE TRYPANOSOMIASIS (TRYPANOSOMA EVANSI)[1]

By RAYMOND RANDALL

Major, Veterinary Corps, United States Army
Member, United States Army Medical Department Research Board
Bureau of Science, Manila

The manner in which pathogenic trypanosomes produce death in their hosts has been a subject of considerable controversy. One method of approach in an attempt to solve the problem, which has been utilized by many workers, is by the study of the alterations of the blood chemistry in experimental trypanosomiasis.

Opposing views on the lethal factors in death from trypanosomiasis have been set forth as follows: First, that the injury and ultimate death of the host are due to a toxic substance liberated by the disintegration of the parasites, as proposed by Regendanz and Tropp (1927) and others; second, that the cause of injury and ultimate death of the host is the exhaustion of the blood sugar and glycogen reserve, as held by Schern (1925–28) and von Fenyvessy (1926); third, in rats at least, Andrews, Johnson, and Dormal (1930) believe that death of the host is due to asphyxia brought about by pulmonary œdema due to partial obstruction of the circulation by the agglutination of the trypanosomes in the heart and lungs.

Regendanz and Tropp, who support the toxin theory, were unable to confirm the findings of Schern and von Fenyvessy in regard to the exhaustion of blood sugar and glycogen reserve. Their results indicate that the glycogen depletion is by no means complete and at the height of the infection sufficient glycogen remains in the liver to maintain a normal sugar concentration in the blood. They maintain that the decrease in the blood sugar is due to a depressive effect on sugar inversion by the trypanosome toxin and not the depletion of glycogen.

[1] This paper was read in part before the Fifteenth Annual Meeting of the Philippine Veterinary Medical Association February 11, 1933, and the completed manuscript submitted for publication October, 1933.

281566—7

Kligler, Geiger, and Comaroff (1929), investigating von Fenyvessy's toxin theory, injected massive doses of autolyzed trypanosomes and plasma respectively from guinea pigs heavily infected with various pathogenic trypanosomes into rabbits and rats, the animals receiving from seven to ten injections in the course of two weeks. In the rabbits the injection of autolyzed trypanosomes and, to some extent, the injection of plasma alone produced a leucopenia. As no other toxic manifestation was observed in either the rats or rabbits, they conclude that the repeated injection of heavy doses of autolyzed trypanosomes did not exert an appreciable toxic effect on the animals. The effect produced, as shown by the leucopenia, is not sufficient to account for the severe damage and ultimate death caused by the infection on the basis of a toxæmia. These workers also made quantitative determinations of the lactic acid in the blood and of the oxygen consumption of rats infected with *Trypanosoma evansi*. They did not find a higher oxygen consumption in infected rats and assume that incompletely oxidized metabolic products of glucose from the activity of the trypanosomes produce a state of acidosis in the animal, leading to exhaustion of the alkali reserve and probably also to a depression of the oxidative processes by the specific effect of lactic acid on the hæmoglobin. By supplementing the alkali reserve twice daily with 0.5 cc of 10 per cent sodium bicarbonate solution, intraperitoneally, they were able to prolong the life of a treated set of rats 50 per cent over that of an untreated set.

Scheff (1928, 1932) agrees that trypanosomes utilize large amounts of glucose and states that the glycogen reserve of the liver becomes exhausted. In guinea pigs, in which the course of infection is prolonged and undulating as compared to the rat, the resulting hypoglycæmia is apparently compensated as shown by a hyperlipemia. He describes in rats infected with *Trypanosoma equiperdum*, a form of internal asphyxia further embarrassing the liver, which is followed by an acidosis contributing to the metabolic disturbances leading to the death of the animal.

Andrews and his associates, using rats infected with *Trypanosoma equiperdum*, state that the animals die of asphyxia and on pathologic evidence, conclude that this is due to a retardation in the circulation of the blood due to agglutination of trypanosomes in the heart and lungs. The consequent anoxæmia leads to a nonvolatile uncompensated acidosis and to central necrosis of the liver, interfering with both its glycogenic and glycogenolytic functions and ultimately producing a hypoglycæmia.

That the mere presence of trypanosomes in a host produces acidosis appears to be disproved by the work of Linton (1930), who found that the nonpathogenic trypanosome, *Trypanosoma lewisi,* in contrast to pathogenic trypanosomes, can be present in the rat in great numbers and yet not produce an acidosis or lowering of the glycogen content of the liver.

The above-described experiments usually have been conducted with small laboratory animals, chiefly rats, an exception being the experiments of Tubangui and Yutuc (1931), who included horses in their studies on the resistance and the blood sugar of animals infected with *Trypanosoma evansi.* These workers conclude that no evidence was obtained to show that resistance against surra is dependent on the ability of the host animal to maintain a normal blood-sugar content. In the majority of their cases, which included rats, guinea pigs, cats, dogs, and horses, no appreciable changes in the sugar content of the blood was observed during the course of the infection except at the very end, when a terminal or agonal hypoglycæmia was usually detected. Their findings were in accord with those of Zotta and Radacovici (1929, *a, b*) who call this condition a premortal hypoglycæmia and state that it occurs not only in trypanosomiasis but also in several other diseases.

Wormall (1932), in determinations on the blood-sugar changes in human trypanosomiasis, found no general hypoglycæmia during the earlier stages of the disease, but at times found values significantly lower than the lowest value with a normal human. Treatment with Bayer 205 gave variable results in relation to the blood-sugar level; sometimes there was a fall, sometimes a rise, and in other cases no change in the blood-sugar level. The injection of 1 gram of Bayer 205 to a patient with the lowest blood-sugar value resulted in a speedy return to normal (or perhaps slightly high) values. He concludes that in glucose tolerance tests carried out on a few of the patients and some of the controls do not suggest that in trypanosomiasis there is a very marked impairment of the capacity of the liver to deal with glucose.

There is very little information available in regard to the blood chemistry changes in the natural hosts of *Trypanosoma evansi.* In the writer's experiments, one of the hosts, the horse, was used with a view to obtaining more information as to the lethal factors in *Trypanosoma evansi* infection (surra) with particular reference to those factors which manifest themselve by changes in the chemical constituents of the blood.

The animals were stabled in screened stalls at the Insular Veterinary Laboratories, Pandacan, Manila. The trypanosome used for inoculation was a strain of *Trypanosoma evansi* that had been isolated from a naturally infected horse by Dr. T. Topacio, of the same laboratories. To prevent an acute explosive type of infection, subcurative doses of Bayer 205 (naganol) were used in amounts that were successful in producing a chronic form of this disease.

The following blood chemistry determinations were made: The carbon dioxide capacity of the blood plasma, and milligrams per 100 cc of blood of nonprotein nitrogen, blood sugar, creatinin, lipoid phosphorus and calculated lecithin. Complete blood and trypanosome counts were also made. The blood was drawn from the jugular vein into flasks containing potassium oxalate and, in addition, neutral paraffin oil was added to the specimens to be used for the carbon dioxide determinations.

To test the theory that the disintegration of trypanosomes liberates a toxin, horse A was infected with *Trypanosoma evansi* and when its blood contained an average of 2,200 trypanosomes per cubic millimeter, it was given 3 grams of Bayer 205 (naganol) intravenously in 60 cc of distilled water. The blood chemistry findings in this horse previous to the administration of the drug were as follows: CO_2 combining power, 57; lipoid phosphorus, 11.40; calculated lecithin, 285; nonprotein nitrogen, 43.5; creatinin, 1.7; temperature, 39.9° C. At the time when the Bayer 205 was given breathing was labored and the animal showed marked ventral swelling and was in a state of clinical acidosis. Twenty-four hours after the administration of Bayer 205, an examination of concentrated portions of blood failed to reveal trypanosomes and inoculation into rats was negative. Clinical symptoms of acidosis were markedly improved and the temperature was 38.4° C. Forty-eight hours after, there was no evidence of clinical acidosis and the temperature had returned to normal. Seventy-two hours after the administration of Bayer 205, blood chemistry determinations showed the following: CO_2 combining power, 68; lipoid phosphorus, 8.30; calculated lecithin, 207; nonprotein nitrogen, 26.6; creatinin, 1.6; temperature and respirations normal. Although the temperature was elevated slightly the next day and remained so for four days, when it returned to normal, trypanosomes during this period could not be demonstrated. The ventral swelling had disappeared on the

fifth day. Had there been an endotoxin liberated by the disintegrating trypanosomes, this, together with the slight toxic action of the drug in an infected animal, should have produced more definite symptoms of a toxæmia. As the blood chemistry findings had returned to normal for the animal, there appears to be no evidence in this case that the death of a large number of trypanosomes in the blood circulation liberated an endotoxin detrimental to the animal.

An attempt was also made to determine the toxic effect of injecting into normal animals a suspension of livers and spleens, rich in trypanosomes, that had been obtained from rats in the terminal stages of trypanosomiasis. Rats were infected with *Trypanosoma evansi* and when in the terminal stages of infection were destroyed. Immediately the organs were removed, weighed, then ground to a fine pulp and suspended in twice their weight of sterile physiological salt solution, with the addition of 1 per cent chloroform for a preservative and to kill the trypanosomes. Two normal horses were given this suspension subcutaneously in three doses at four-day intervals in the following amounts: 3 cc, 5 cc, and 10 cc. Other than local swelling that subsided in the interim, there was no clinical evidence of a toxic condition being produced by these injections. The injections made the animals hypersensitive to a later inoculation of live trypanosomes, as was shown by a shorter incubation period, as compared to other animals not so treated.

Primarily to determine if death is due to an exhaustion of blood sugar and to measure the amount of blood sugar present during the infection and at time of death, horses B and C were inoculated with *Trypanosoma evansi.* Results of blood chemistry and blood picture findings during the course of the disease are given in Table 1. By the use of small doses of Bayer 205 (naganol) the disease ran a chronic course in both animals with many crises and relapses. As the infection progressed a marked anæmia developed. Acidosis was more marked at the time of crises at which times the animals had a lower blood-sugar content, with a higher content of lipoid phosphorus, calculated lecithin, nonprotein nitrogen, and creatinin. Shortly following these crises, the CO_2 combining power of the plasma increased, but never returned to normal, and the lipoid phosphorus, calculated lecithin, nonprotein nitrogen, and creatinin showed a decrease. The blood-sugar level usually rose after a crisis and was at times higher than normal.

The average weight of the horses at time of inoculation was 1,000 pounds. Horse B lived for seventy days and horse C lived for forty-four days after inoculation, and the weight of each of the animals had decreased approximately 25 per cent at the time of death.

TABLE 1.—*Blood changes in horses infected with Trypanosoma evansi.*

Period of infection.	Volume per cent. Carbon dioxide capacity.	Milligrams per 100 cc of blood.					Number per cemm of blood.		
		Lipoid phosphorus.	Lecithin.	Nonprotein nitrogen.	Creatinin.	Sugar.	Red blood cells. Millions.	White blood cells. Thousands.	Trypanosomes. Thousands.
HORSE B.									
Before infection	70.0	8.5	212	25.5	1.5	85	6.9	5.5	0
Thirty-five days after infection.	52.3	11.8	295	33.3	1.8	87	3.8	10.5	45
Forty-eight hours prior to death.	45.8	13.3	332	50.0	1.8	72	3.9	7.7	20
Twenty-four hours prior to death.	30.0	14.3	357	54.6	2.3	27	5.5	12.8	102
One-half hour prior to death.	18.3	20.0	500	108.0	2.0	71	5.3	12.4	70
HORSE C.									
Before infection	71.8	8.5	212	33.3	1.6	------	------	------	0
Four days after infection.	74.5	9.5	237	27.8	1.5	88	6.9	6.4	0
Twenty-eight days after infection.	57.9	10.5	262	25.0	1.6	105	2.7	5.9	18
Forty-eight hours prior to death.	52.3	12.2	305	29.0	2.0	95	3.0	8.6	—1
At death	12.1	14.3	357	109.0	3.3	74	------	------	100+

Terminal determinations were made on unclotted blood collected from the ventricles of the heart in the case of horse C at death and one-half hour prior to death in the case of horse B. The increase in both red and white cell counts in the terminal stages of trypanosomiasis is assumed to be due to dehydration of the animals. The CO_2 combining capacity of the blood plasma being 18.3 and 12.1 for horses B and C, respectively, showed that the animals had marked terminal acidosis. The nonprotein nitrogen and creatinin determinations indicated that nephrosis was advanced. In horse C the blood sugar was at no time markedly decreased, but horse B had a blood sugar

of only 27 twenty-four hours previous to death, which had increased to 71 as shown by the specimen obtained only one-half hour before death, while the animal was in a moribund condition. In both cases the urine collected at autopsy was strongly positive for albumin.

Specimens of tissue were obtained for pathological examination, a report on which is not as yet available.

DISCUSSION

The foregoing investigations have not established without doubt the mechanism of death in horses infected with *Trypanosoma evansi.* The results obtained support the theory that death is due to an asphyxia, although the factors leading to this condition are not clearly demonstrable.

It should be noted that in these horses, hypoglycæmia at time of death was not marked. The maintenance of an approximate normal blood sugar level may be explained by the lecithin content of the blood during the infection. It is known that fat circulates in the blood as a lecithinlike compound, and the production of sugar from fat, and of dextrose from glycerol, in the animal body, appears to be possible (Linton, 1930). At time of death, in the cases reported, body fat was almost absent other than a fair portion in the liver. In these horses the appetite, with certain exceptions for short periods, remained normal throughout the course of the disease.

The animals were continuously in a state of acidosis that varied at times according to the number of trypanosomes present in the blood stream. In the determinations made after a crisis, there was a marked scarcity of trypanosomes, the CO_2 capacity of the blood plasma and the blood sugar increased, and the latter at times was above normal. It appears that pathogenic trypanosomes use glucose to an extent that causes withdrawal of the body fat to the blood stream as lecithin, which is in turn converted into glucose. The abnormal blood sugar at times after a crisis indicates that conversion goes on at a rate that takes some time to stabilize and by this conversion the body is able to maintain an approximately normal sugar level until death approaches. The end products of glucose consumption cause an uncompensated acidosis and exhaustion of the alkali reserve, which is further adversely affected by the destruction of the red cells causing an anoxæmia that manifests itself by an ac-

celerated and labored respiration in the infected animal. As the animals reached the terminal stage of infection it was noted that there was an increase in both red and white cell counts, which may be assumed to be due to a terminal dehydration of the animal.

There is evidence that trypanosomes utilize carbohydrates in their metabolism and some workers believe that a hypoglycæmia may cause the death of trypanosomes in vivo. Whether or not this is a factor in the production of crises in an infected animal or that trypanocidal antibodies are the sole or major factor in the destruction of trypanosomes is not conclusively established. If there is any in vivo destruction of trypanosomes by a hypoglycæmia of the host, it has not been indicated by the findings in the infections with *Trypanosoma evansi* in equines herein reported.

CONCLUSION

It is believed that *Trypanosoma evansi* causes death in equines not by the production of a toxic substance liberated by the disintegration of the organisms, nor by the exhaustion of the blood sugar and glycogen reserve, but by an asphyxia from an uncompensated acidosis, the mechanism of which is still undetermined.

REFERENCES

ANDREWS, J., C. M. JOHNSON, and V. J. DORMAL. Lethal factors in experimental infections of Trypanosoma equiperdum in rats. Am. Journ. Hyg. 12 (1930) 381–400.

VON FENYVESSY, B. Über die Bedeutung des Stoffwechsels der Parasiten für das Wirtstier bei der Trypanosomeninfection. Biochem. Ztschr. 173 (1926) 289–297.

KLIGLER, I. J., A. GEIGER, and R. COMAROFF. Susceptibility and resistance to trypanosome infections. Ann. Trop. Med. and Parasitol. 23 (1929) 325–335.

LINTON, R. W. A comparison of the chemical alterations in the blood of rats infected with pathogenic and nonpathogenic trypanosomes. Journ. Exper. Med. 52 (1930) 695–700.

LINTON, R. W. The blood chemistry of an acute trypanosome infection. Journ. Exper. Med. 52 (1930) 103–111.

REGENDANZ, P., and C. TROPP. Das Verhalten des Blutzuckers und des Leberglykogens bei mit Trypanosomen infizierten Ratten. Arch. f. Schiffs.- u. Trop.-Hyg. 31 (1927) 376–385.

SCHEFF, G. Über den intermediären Stoffwechsel der mit Trypanosomen infizierten Ratten. Biochem. Ztschr. 200 (1928) 309–330.

SCHEFF, G. Über den intermediaren Stoffwechsel der mit Trypanosomen infizierten Meerschweinchen. Biochem. Ztschr. 248 (1932) 160–180.

SCHERN, K. Über Trypanosomen. Centrabl. f. Bakt. 96 (1925) 356–365; 440–454.

SCHERN, K. Über die Störung des Zuckerstoffwechsels bei Trypanosomiasen und Spirochätosen. Biochem. Ztschr. 193 (1928) 264–268.

TUBANGUI, M. A., and L. M. YUTUC. The resistance and the blood sugar of animals infected with Trypanosoma evansi. Philip. Journ. Sci. 45 (1931) 93–105.

WORMALL, A. Carbohydrate metabolism in human trypanosomiasis. Biochem. Journ. 26 (1932) 1777–1787.

ZOTTA, G., and E. RADACOVICI. Contribution à l'étude du métabolisme du glucose sanguin la trypanosomiase expérimentale. Arch. Roumaines Path. Expérim. et Microbiol. 2 (1929) 55–80.

ZOTTA, G., and E. RADACOVICI. Sur les variations de la glycémie dans la trypanosomiase. C. R. Soc. Biol. 102 (1929) 129–130.